CARDIOVASCULAR MANIFESTATIONS IN LEPTOSPIROSIS, MALARIA AND DENGUE- A STUDY OF 100 CASES

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APRIL 2011

CERTIFICATE

This is to certify that the dissertation titled **"CARDIOVASCULAR MANIFESTATIONS IN LEPTOSPIROSIS, MALARIA AND DENGUE- A STUDY OF 100 CASES"** is the Bonafide original work of **Dr. PRASANTH SANKAR** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in **APRIL 2011**. The Period of study was from OCTOBER 2009 to OCTOBER 2010.

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DECLARATION

I, **DR. PRASANTH SANKAR ,** solemnly declare that dissertation titled **"CARDIOVASCULAR MANIFESTATIONS IN LEPTOSPIROSIS, MALARIA AND DENGUE- A STUDY OF 100 CASES"** is a Bonafide work done by me at Govt. Stanley Medical College and Hospital during OCTOBER 2009 to OCTOBER 2010 under guidance and supervision of my unit chief **Prof. G. ELANGOVAN M.D**.

This dissertation is submitted to Tamil Nadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine.**

Place : Chennai Dr. Prasanth Sankar

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CONTENTS

INTRODUCTION

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Leptospirosis, Malaria and Dengue are the leading causes of acute febrile illness in both in-patients and out patients in Chennai. Though caused by a bacterium, a virus and a protozoan, and transmitted by different agents, the heavy rainfall, water stagnation and poor drainage facilities in Chennai unite these three agents under a single umbrella. They are highly prevalent in places from North Chennai like Tondaiarpet, Washermanpet, Royapuram, Harbour, Mannady, Pattalam & Pulianthope.

The cardiovascular manifestations seen in patients with Leptospirosis, Malaria and Dengue are well documented in literature but have rarely been reported in the numerous clinical and epidemiological studies done in Chennai. These findings are often considered nonspecific or secondary manifestations of fever and are overlooked during the routine evaluation of the infectious disease. But world literature clearly shows the myocardial involvement in these diseases.

Cardiac involvement can be due to direct invasion, immune mediated or as a response to systemic changes. The subtle clinical and investigational findings may at times predict underlying cardiac involvement and may be markers of life threatening complications. The problem is glorified in the population of Chennai who are prone to develop co infection with two of these agents or even three agents together.

Cardiac involvement in leptospirosis is common and is often underestimated. Clinical evidence of myocardial involvement comprises myocarditis and abnormal T wave changes, RWMA, chamber dilatation in 2D echo. Repolarization abnormalities and arrhythmias on ECG were considered poor prognostic indicators in severe leptospirosis cases.

Dengue is known to affect several systems in the human body. Acute reversible myocardial dysfunction is the commonest documented cardiac complication. Dengue myocarditis is generally reversible with favorable outcomes if diagnosed and treated early. Alternation of

autonomic tone, rhythm disorders, such as atrioventricular blocks and ventricular ectopic beats, ST segment and T wave changes, low ejection fractions, and global hypokinesia on radionuclide ventriculography have been reported.

Cardiac complications in malaria have been infrequently reported. Transient but clinically significant ECG & 2D echo abnormalities have been documented in acute stages of malaria.

Myocarditis has been documented in severe malaria due to both P. vivax and P. falciparum.

Most interesting are the reports from various hospitals in Mumbai, where they have reported 8 cases of myocardial infarction, left ventricular dysfunction and stroke in young patients with no cardiovascular risk factors, during the course of vivax malaria due to a suspected mutated strain.

This study was undertaken at Stanley Medical College Hospital which is located in North Chennai. Here an attempt is made to assess and compare the cardiovascular manifestations and outcome in Leptospirosis, Malaria and Dengue.

AIM OF THE STUDY

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- To study the various cardiovascular manifestations and their outcome in patients with Leptospirosis, Malaria and Dengue.

OBJECTIVES

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1. To study the clinical profile like age, sex, fever duration and associated cardiovascular symptoms in patients with Leptospirosis, Malaria and Dengue.
2. To compare and contrast the various cardiovascular manifestations in these patients.
3. To compare and contrast the different laboratory parameters in these patients.
4. To assess the relationship between various clinical, electrocardiographic and echocardiographic findings in these patients.
5. To assess the outcome of the illness in the patients with co- infection.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

LEPTOSPIROSIS

Leptospirosis has been under reported and under diagnosed from India due to a lack of awareness of the disease and lack of appropriate laboratory diagnostic facilities in most parts of the country. Cases have been reported from Kerala, Tamil Nadu, Gujarat, Andaman, Karnataka, Maharashtra, Orissa and Bihar. [1,2]

Leptospirosis has been reported from Chennai since 1980's.[6,7] The leptospirosis laboratory at the Institute of Microbiology, Madras Medical College was established in 1994. This laboratory receives samples from both government and private hospitals. Data on leptospirosis from government hospitals during the period 2004 – 2006 is given in table 1.[3,8]

Table 1

Year	**2004**	**2005**	**2006**
Leptospirosis	**963**	**1724**	**2765**

There has been a dramatic increase in the number of leptospirosis cases in the year 2006 during which 2765 cases were reported.

Leptospirosis is an important cause of acute febrile illness in Chennai. In a recent study of 500 cases of fever at government Stanley hospital, leptospirosis was the second common cause of fever contributing to 17%, following malaria which was 27%. Coinfection of leptospirosis (48 cases) with malaria (220 cases) occurred in 22% of cases.[9,10] Leptospirosis can manifest in many ways.[6] The various syndromes of presentation are as follows.

1. Acute febrile illness
2. Weil's syndrome characterized by jaundice, renal failure and myocarditis.
3. Pulmonary Haemorrhage with respiratory failure
4. Meningitis / Meningoencephalitis

The incubation period is 7—14 days, but ranges from 2—21 days.

In a recent study from Chennai, Fever (98.6%), Myalgia (72.9%), headache (48.6%), Conjunctival suffusion (38.8%), meningism (2.7%) and jaundice (5.5%) were the common clinical manifestations.[8] The Modified Faine's Criteria (Table 3) is utilized for diagnosis of leptospirosis in district / teaching institutes.[5,11]

Table 3

Modified Faine's criteria

Clinical features (A)	**Score**	
Fever	**2**	
Headache	**2**	
Temperature > 39 deg.C	**2**	
Myalgia	**4**	
Conjunctival suffusion	**4**	**10**
Meningism	**4**	
Jaundice	**1**	
Albuminuria/ elevated BUN	**2**	
Epidemiological factors (B)		
Rainfall	**5**	
Contaminated environment	**4**	
Animal contact	**1**	
Laboratory criteria (C)		
Culture	**Diagnosis certain**	
ELISA IgM	**15**	
MSAT	**15**	
MAT- single positive high titer	**15**	
MAT- rising titer (paired sera)	**25**	

Presumptive diagnosis of leptospirosis is made of:

- *Part A or part A+B with a score of 26 or more*
- *Part A+B+C = 25 or more and in serological tests, only one test should be scored.*

A score between 20 and 25 suggests leptospirosis as a possible diagnosis.

Previous studies of patients with severe leptospirosis have usually focused on the hepatic and renal manifestations of this illness. Despite the fact that many patients may have associated cardiac involvement, these manifestations are seldom discussed in reviews on the clinical aspects of this disease.[12,13]

Studies by Shiva Kumar et.al. suggested that though incidence of severe Leptospirosis has declined, mild Leptospirosis has increased.[3,8] The reasons for decline of severe Leptospirosis suggested were greater awareness of the disease, availability of better diagnostic facilities and widespread use of antibiotics. In addition, serogroup Autumnalis, a virulent serogroup causing severe Leptospirosis has also declined since 1995. The increase in mild Leptospirosis suggests that the environmental risk factors (Infected rodents and domestic animals, contaminated environment and rainfall) play an important role in the persistence and spread of the disease.[3,8]

Cardiac involvement in leptospirosis is common but may be underestimated. Clinical evidence of myocardial involvement including abnormal T waves was detected in 10 % of 80 severe icteric cases in Lousiana. While similar ECG abnormalities were detected in over 40 % of patients in China, India, Sri Lanka, and the Philipinnes including both icteric and non icteric cases.[15,16,17]

In 1951 Sodeman and Killough reported data from 80 patients with leptospirosis,of whom 8 (10%) had definite evidence of cardiac involvement.[12]

In a study conducted in Gujarat, Out of twenty-five seropositive patients, 14(56%) had cardiovascular manifestations. Electrocardiography abnormalities were seen in 13(52%) patients. The commonest finding was first-degree AV block seen in 11(44%) patients followed by ST-segment depression in four (16%) patients, T-wave inversion in leads II, III and avF in two (8%) patients, corrected QT-interval prolongation in three (12%) patients and ventricular premature beats in two (8%) patients. Atrial fibrillation was seen in only one patient. Left ventricular function as assessed by two-dimensional echocardiography was normal in all patients.[17]

An analysis of data of 50 patients with serologically proven leptospirosis demonstrated that 70% of the patients had electrocardiographic abnormalities, with atrial fibrillation being the

commonest major arrhythmia noted. Thirty-six percent of the patients had conduction system abnormalities and 30% had T wave changes. [18] Another series has reported AV block in 44% of patients with leptospirosis.[19] A glycoprotein fraction of leptospiral cell wall has been incriminated in the pathogenesis of these rhythm disturbances. This protein is thought to inhibit the Na-K ATPase and may be responsible for the arrhythmia.[20] Univariate analysis has shown that cardiac arrhythmia is more common in patients dying of leptospirosis than in the survivors.[21] Other reported cardiac abnormalities include myocarditis and pericarditis.[22] In seven cases of fatal leptospirosis, petechial haemorrhages were found in the heart and the pericardium in all the autopsy specimens and interstitial myocarditis was found in five specimens.[23]In another study, acute coronary arteritis was found in 70% of patients who died of leptospirosis and evidence of aortitis was present in more than half.[24] One case of leptospira endocarditis, possibly caused by serovar Icterohaemorrhagiae has been reported in literature.[25]

A recent study from Maharashtra found out Myocarditis in 96% and Endocardial inflammation in 50% on autopsy of patients who had died from leptospirosis. The study concluded that there is definite cardiac involvement in leptospirosis, which even though not symptomatically evident, may add to the morbidity or be contributory to the mortality associated with the disease. In addition, a possibility of dilated cardiomyopathy as a delayed

MALARIA

There are four species of human malarial parasite i.e. Pl.vivax, Pl.falciparum, Pl.malariae and P1.ovale. In India 60 - 65% of infections are due to Pl.vivax and 35 to 45% are due to Plasmodium falciparum.[29] Despite effects at vector control, malaria still remains a major public health problem. Malaria remains to be one of the world's most prevalent infectious diseases. About 300 – 500 million cases are reported annually allover the world with a mortality of about 1.1 to 2.7 million. 90% of these cases are reported from Africa.[29] In India,

2.5 to 3 million cases and 1000 deaths of malaria are reported annually. Many consider this is an underestimate. The parasite profile has been changing significantly over the years with a steady increase in percentage of PF cases across the country (50.5% of cases in 2005). Areas with more than 30% of PF cases are categorized as high risk. These include North East India, Orissa, Jharkand, West Bengal, Madhya Pradesh, Maharashtra & Andrapradesh.[30,31] In Tamil Nadu 70% of malaria cases are reported from Chennai. The Chennai Corporation has recorded more than 1,100 malaria cases during May and June 2010 and more than 2,877 cases during January to June 2010.[46] Malaria is highly prevalent in places from North Chennai like Tondiarpet, Washermanpet, Royapuram, Mannady, Pattalam & Pulianthope.

The clinical features of ***uncomplicated malaria*** are common to all species. Head ache, muscular ache, abdominal discomfort, lethargy and lassitude often precede fever by about 2 days. The fever in Plasmodium vivax and Plasmodium ovale regularises to a two day cycle and in Plasmodium malaria to a 3 day cycle. The fever in Pl. falciparum may never regularise to a classical tertian pattern.

DEFINITION OF SEVERE MALARIA BY WHO[29]

1. Renal failure: Sr.creatinine > 3 mgs / dl, Urine output < 400 ml / 24 hrs (or) < 12 ml / hr
2. Severe Anaemia Hb% < 5 gms % (or) Hct < 15%
3. Cerebral malaria: Unarousable coma with peripheral parasitemia
4. Pulmonary Oedema or acute respiratory distress syndrome
5. Hypoglycemia: Blood sugar < 40 mg / dl
6. Shock - Systolic BP < 70 mm Hg (Adults) < 50 mm Hg (Children - 1-5 yrs)
7. Acidemia - pH < 7.33, Hco3 < 15 mq / l
8. Spontaneous bleeding: Gums, Nose and GIT due to DIC
9. Macroscopic haemoglobinuria
10. Seizures > 3 episodes

11. Hyperparasitemia - More than 20%

12. Jaundice: Serum bilirubin > 3 mgs / dl is a marker of severe malaria, only when combined with other vital organ dysfunction (i.e. cerebral malaria and renal failure).

In a prospective study of malaria at Bombay during June 1993 to May 1994, all patients had fever, followed by head ache (92%), vomitting (74%), cough (7%), diarrhoea (4.6%), icterus (3.8%) and oliguria (1%) 70% were due to Plasmodium vivax and 30% due to Pl.falciparum.[32] Mohapatra et al.,from Berhampur has reported various atypical presentation of malaria in their study of 110 cases of vivax malaria. They were absence of malarial paroxysm (22.8%), migrainous head ache (4.5%), myalgia (6.3%), episodic utricarial rash (1.8%), relative bradycardia (13.6%) and postural hypotension (2.7%).[34]

In a recent study from Bikaner, Kochar et al. reported 11 cases of definite severe vivax malaria. In all these cases Plasmodium falciparum infection was ruled out by PCR. All other infectious causes were ruled out by appropriate tests. In this study the manifestations observed were jaundice and hepatic dysfunction in four, severe anaemia in four, cerebral malaria in three, ARDS in three, bleeding diathesis in one and MODS (multi organ dysfunction syndrome) in five patients.[36]

Cardiac involvement in malaria has not been studied widely. Myocardial function is generally well preserved in severe falciparum malaria. There have been few reports of experimental and postmortem studies indicating myocardial involvement in malaria.[41,42] The cardiac index may be elevated with low peripheral vascular resistance and low-to-normal ventricular filling pressures. Hypovolaemia also may contribute to the reduced pressures. There may be reduction in visceral perfusion. Orthostatic hypotension is common. Septicaemia, metabolic acidosis and hypoxia may result in a drop in cardiac index. Malaria can also complicate pre-existing cardiac decompensation and may even prove fatal for patients with compromised heart. Cardiac arrhythmias are uncommon.[37] A variety of cardiac arrhythmias especially due

to prolongation of QT interval have been reported frequently by the use of drugs like quinine, quinidine and mefloquine. Complete AV dissociation was observed in a case which could be due to myocardial ischaemia caused by sequestration of red cells in the coronary circulation.[39]

A retrospective observational study of 38,919 in-patients of Dr. TMA Pai Rotary Hospital, Mangalore, was done from the year 1995 to 1998, and it was found that among 1531 malarial patients, 22 had AMI (1.43%), a statistically significant ($P < 0.05$) occurrence, as compared to AMI among all in-patients who were in for complaints other than malaria, (0.82%), reflecting the possibility of myocardial damage in malaria.[40]

In a study of 22 patients without a previous history of cardiac disease, during the acute phase ECG abnormalities were common (5/22); pericardial effusion was found in 2 patients and global left ventricular hypokinesia in 1 patient infected with Plasmodium falciparum.[43] Cardiac involvement and myocardial dysfunction has also been noted in childern with severe falciparum malaria.[44] A case of myocarditis associated with *P* vivax infection in a 27-year-old woman has been reported.[45]

In Mumbai, over 8,600 cases of malaria were reported in the first 17 days of July and also 2 people died. There are reports of mutated strains of plasmodium vivax causing myocardial infarction. At least 8 cases of myocardial infarction have been reported. Angiography in some of the cases has demonstrated clots in coronary arteries, with the patients having no conventional risk factors for heart disease. Patients who survived showed improvement in LV function with anti-malarial treatment.[47]

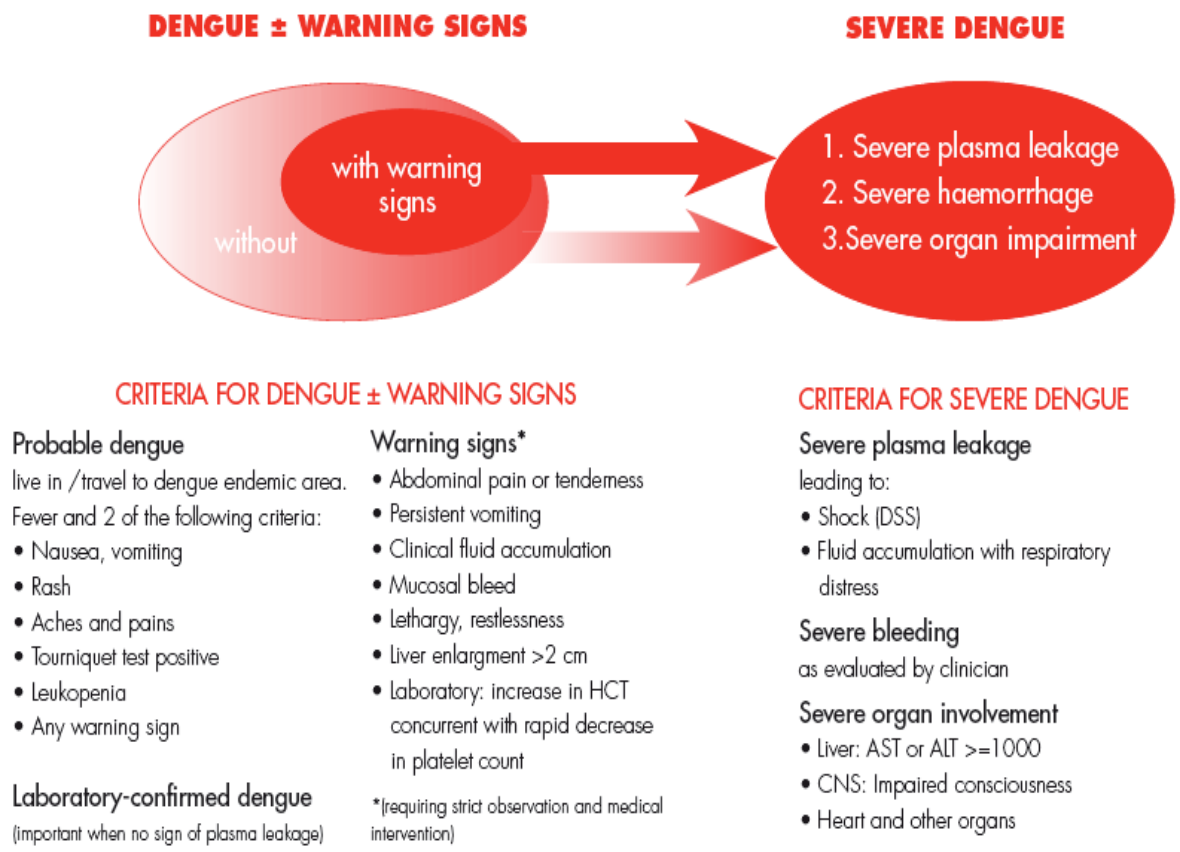
DENGUE

Dengue is the most rapidly spreading mosquito-borne viral disease in the world. In the last 50 years, incidence has increased 30-fold with increasing geographic expansion to new countries and, in the present decade, from urban to rural settings. The 2005 World Health Assembly

resolution WHA58.3 on the revision of the International Health Regulations (IHR) includes dengue as an example of a disease that may constitute a public health emergency of international concern with implications for health security due to disruption and rapid epidemic spread beyond national borders. In 2003, eight countries -- Bangladesh, India, Indonesia, Maldives, Myanmar, Sri Lanka, Thailand and Timor-Leste -- reported dengue cases. Reported case fatality rates for the region are approximately 1%, but in India, Indonesia and Myanmar, focal outbreaks away from the urban areas have reported case-fatality rates of 3--5%. In India, 5,534 cases of Dengue were reported in 2007 with a mortality of 80 cases. Case Fatality Rate in 2007 was 1.25%. In 2008, 12,419 cases of Dengue were reported with a mortality of 69 cases. Case Fatality Rate was 0.6%. In 2006, Tamilnadu contributed to 4 % (477) of cases, most of which are from Chennai. No deaths were reported. Dengue afflicts all levels of society but the burden may be higher among the poorest who grow up in communities with inadequate water supply and solid waste infrastructure, and where conditions are most favourable for multiplication Ae. aegypti.[49]

Expert consensus groups in Latin America (Havana, Cuba, 2007), South-East Asia (Kuala Lumpur, Malaysia, 2007), and at WHO headquarters in Geneva, Switzerland in 2008 agreed that: "*dengue is one disease entity with different clinical presentations and often with unpredictable clinical evolution and outcome*"; the classification into levels of severity has a high potential for being of practical use in the clinicians' decision as to where and how intensively the patient should be observed and treated, in more consistent reporting in the national and international surveillance system, and as an end-point measure in dengue vaccine and drug trials.[49,50]

Suggested dengue case classification and levels of severity:



This model for classifying dengue has been suggested by an expert group (Geneva, Switzerland, 2008) and is currently being tested in 18 countries by comparing its performance in practical settings to the existing WHO case classification. The process will be finalized in 2010.

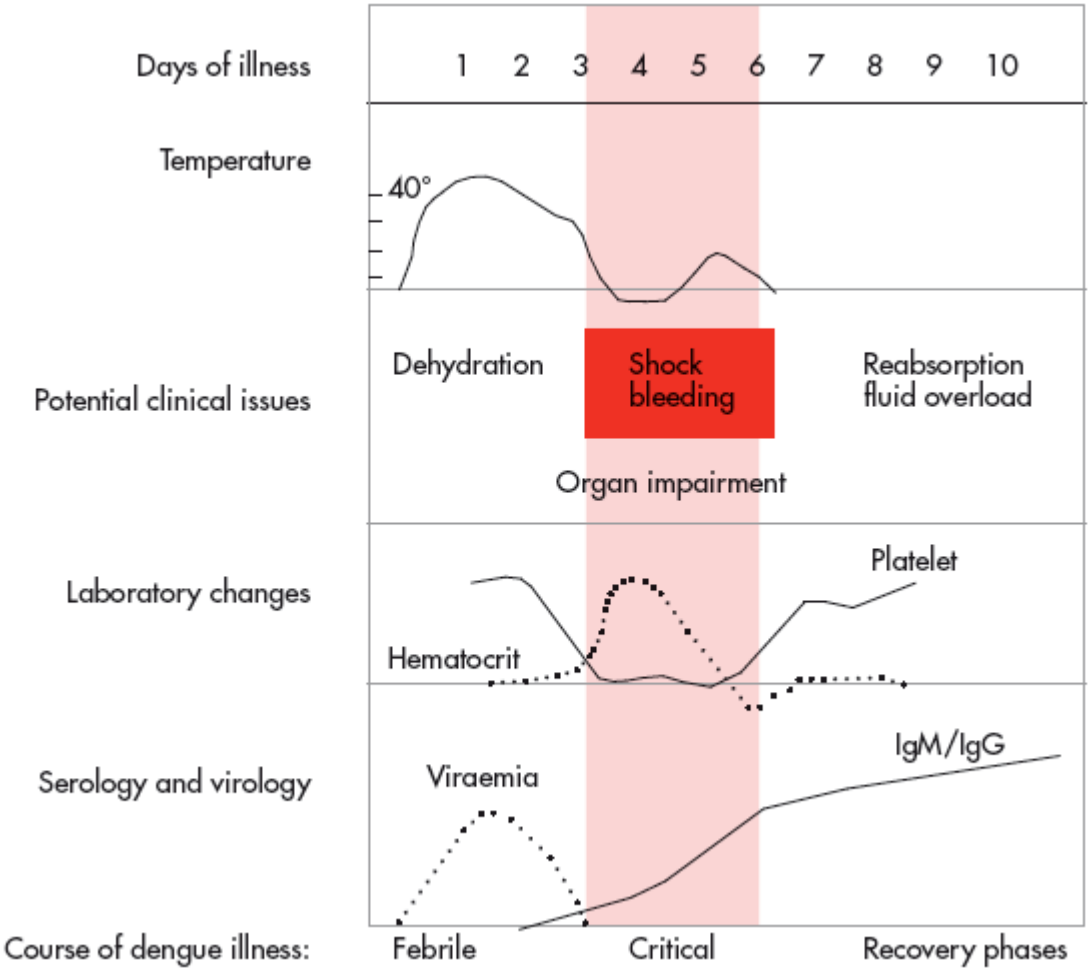
For practical reasons this guide adapts the distinction between dengue and severe dengue.[49,50]

The various clinical problems during the different phases of dengue can be summarized as in Table 4 and Figure 1

Table 1

1	Febrile phase	Dehydration; high fever may cause neurological disturbances and febrile seizures in young children
2	Critical phase	Shock from plasma leakage; severe haemorrhage; organ impairment
3	Recovery phase	Hypervolaemia (only if intravenous fluid therapy has been excessive and/or has extended into this period)

Figure 1



There is little published data on myocardial involvement in acute dengue infection. Myocarditis is the most common documented cardiac pathology in dengue, however, only a few cases are reported in the world literature. Wiwanitkit proposed a probable reason for the low incidence of dengue myocarditis, that it might represented the rarity of the cases or it might be due to underdiagnosis and neglecting to report.[51] Wiwanitkit also concluded that such myocarditis was very rare and might not be fatal if early diagnosed and treated.[51] However, in another report from Columbia, there was a high incidence of myocarditis.[52] Concerning the manifestation of dengue myocarditis, Horta Veloso et al, reported that cardiac rhythm disorders, such as atrioventricular blocks and ventricular ectopic beats, can appear during infection and are attributed to viral myocarditis.[53] Dengue endocarditis has never been reported. Indeed, the formed immune complex in dengue infection could not be entrapped in

the valvular space; therefore, dengue endocarditis could not exist. Dengue pericarditis can be seen but it is very rare and in the form of myopericarditis.[54] The pathogenesis is believed to be the extension of dengue myocarditis into the pericardium rather than circulating immune complex. At present, dengue pericarditis is extremely rare due to early diagnosis of dengue myocarditis.

Myocardial involvement may be a result of the direct effect of the dengue virus in susceptible individuals, or due to the effects of cytokine mediators and/or cellular components of the immune response. The possibility of IgM antibodies produced against the dengue virus cross-reacting with a myocardial antigen is unlikely, as echocardiographic improvement was seen within three weeks of the illness, when these antibodies were still in circulation. Dengue viral antigen, associating with a myocardial receptor site, thereby triggering off an immunological response is also another possibility in susceptible individuals. This myocardial inflammation ceases when the viral antigen disappears from the circulation. Dengue haemmorrhagic fever patients have higher levels of TNF-α, interleukins- 6, -13 and -18, and cytotoxic factor. These cytokines are implicated in causing increased vascular permeability and shock during dengue infection but their contribution towards development of myocarditis remain undefined.[55,56,57]

In an Indian study, Wali *et al.* described cardiac involvement in dengue haemorrhagic fever/dengue shock syndrome (DHF/DSS) in 17 patients, which showed global hypokinesia in 70.6%. Tc^{99} pyrophosphate imaging carried out in four patients showed no myocardial necrosis. Only five patients showed ST and T wave changes with ECG changes, echocardiographic and radionuclide ventriculography all returning to normal within three weeks. The results of a study from Sri Lanka revealed that myocardial involvement of dengue infections run a benign course without long-term complications, contrary to what was believed in the past. Evidence of 2-D echocardiographic myocarditis was seen in 24%. Male to female ratio was 2:1 while 65% were in 12–30 years age group.[59] Three cases of DEN 3 who

had significant cardiac dysfunction suggestive of myocarditis were reported in an outbreak of dengue fever in Kandy, Sri Lanka in April 2005.[60]

In a study of 11 children with DHF who presented with symptoms of myocarditis, widespread viral infection of the myocardial endothelium, and cardiomyocytes, accompanied by inflammation was observed in 1 fatal case. Myocytes were infected by dengue virus and had increased expression of the inflammatory genes and protein IP-10. Infected myocytes had increased intracellular calcium concentration, which may directly contribute to the presentation of myocarditis in pediatric patients.[61]

In a retrospective study done on 28 patients with DHF, none had clinical features of overt myocarditis. Five patients (17.8%) had sinus bradycardia (HR<60 bpm,) and there were no other ECG abnormalities. Two patients (71%) had significantly raised cardiac enzymes CPK-MB, LDH and SGOT. Twelve patients (42.8%) were positive for Serum Troponin-T. Two patients (7%) had grade-1 diastolic dysfunction in 2D-ECHO and one patient (3.5%) had mild pericardial effusion.[62]

Myocarditis is clinically and pathologically defined as "inflammation of the myocardium." Despite its rather clear-cut definition, the classification, diagnosis, and treatment of myocarditis continue to prompt considerable debate. Clinical presentations of the disease range from nonspecific systemic symptoms (fever, myalgias, palpitations, or exertional dyspnea) to fulminant hemodynamic collapse and sudden death. The extreme diversity of clinical manifestations has made the true incidence of myocarditis difficult to determine.[80]

Despite the well-established morbidity and mortality associated with myocarditis, clinical practice guidelines with regard to its evaluation and treatment are lacking. The wide variety of etiologies implicated in myocarditis and its heterogeneous clinical presentations have impeded patient identification and consensus on the most appropriate diagnostic criteria. The

Dallas pathological criteria, published in 1986, served as the first attempt to develop standardized diagnostic guidelines for the histopathological classification of myocarditis.[80,83] Clinical manifestations range from asymptomatic ECG abnormalities to cardiogenic shock.[85] Transient ECG abnormalities suggesting myocardial involvement commonly occur during community viral endemics; most patients remain entirely asymptomatic. In contrast, myocarditis can also result in fulminant heart failure presenting as new-onset cardiomyopathy. Patients may report a viral prodrome of fever, myalgias, respiratory symptoms, or gastroenteritis followed by an abrupt onset of hemodynamic collapse. The incidence of a reported infectious viral prodrome is highly variable, ranging from 10% to 80% of patients with documented myocarditis.[86] Acute dilated cardiomyopathy is one of the most dramatic and clinically relevant presentations of acute lymphocytic myocarditis. The link between clinical myocarditis and acute dilated cardiomyopathy is most convincingly provided by EMB findings.[85] The 2 largest biopsy series have confirmed myocarditis in 9% to 16% of cases of new-onset dilated cardiomyopathy.[87] The Giant Cell Myocarditis Study Group identified heart failure symptoms as the primary presentation in 75% of patients with giant cell myocarditis. Neither symptoms nor clinical course of myocarditis has been shown to correlate with histopathological features such as the extent of lymphocytic infiltrate or fibrosis. The classification of Lieberman et al[84] differentiates fulminant from active myocarditis. Fulminant myocarditis, manifested by severe hemodynamic compromise requiring high dose vasopressor support or mechanical circulatory support, was identified in 15 of 147 patients (10.2%) in the largest prospective study to use this classification system. Fulminant cases were additionally characterized by a distinct viral prodrome, fever, and abrupt onset (generally ~3 days) of advanced heart failure symptoms. These patients typically have severe global left ventricular dysfunction and minimally increased left ventricular end-

diastolic dimensions. Of note, either borderline or active lymphocytic myocarditis can produce this dramatic clinical presentation.

Myocarditis masquerading as an acute coronary syndrome has also been well described.[88] Elevated troponin levels have proven to be a more reliable predictor of myocardial injury than levels of creatine kinase. ECG changes suggestive of acute myocardial ischemia typically may include ST-segment elevation in 2 contiguous leads (54%), T-wave inversions (27%), widespread ST-segment depressions (18%), and pathological Q waves (18% to 27%).[89] Segmental or global echocardiographic wall motion abnormalities are frequently evident despite angiographically normal coronary anatomy.[89] Sarda et al, using myocardial indium111-labeled antimyosin antibody and rest thallium imaging, identified 35 of 45 patients (78%) who presented with acute chest pain, ischemic ECG abnormalities, and elevated cardiac biomarkers as having myocarditis.[88] However, biopsy verification of actual myocarditis was not undertaken in this series. In another series of 34 patients with known normal coronary anatomy presenting with symptoms and ECG changes consistent with an acute coronary syndrome, 11 (32%) of the patients were found to have myocarditis on biopy. Clinicians should consider acute myocarditis in younger patients who present with acute coronary syndromes when coronary risk factors are absent, ECG abnormalities extend beyond a single coronary artery territory, or global rather than segmental left ventricular dysfunction is evident on echocardiography.

Myocarditis can produce variable effects on the cardiac conduction system. Ventricular tachycardia is an uncommon initial manifestation of myocarditis but often develops during long-term follow-up.[88] The Giant Cell Myocarditis Study Group reported an initial incidence of ventricular tachycardia of 5% in a multicenter cohort. Ventricular tachycardia due to either lymphocytic or granulomatous myocarditis may infrequently result in sudden cardiac death.[90]

The definitive diagnosis of myocarditis has to be established by the demonstration of myocytolysis and the lymphocytic infiltrates in the endomyocardial biopsy (EMB) specimens (Aretz *et al,* 1987). However, EMB has confirmed the diagnosis of myocarditis in only about 10 - 25% of the patients in whom this disease was clinically suspected (Fowles *et al,* 1984; Parrillo *et al,* 1984; Mason *et al,* 1995), and repeated EMBs are not warranted in some clinical situations. During the course of myocarditis, the laboratory markers of myocardial cell damage, such as creatine kinase (CK) and creatine kinase MB isoform (CK-MB) levels are often within the normal range.[93,94] Cardiac isoforms of troponin T or I (cTnT, cTnI) are only expressed in cardiac muscle and their serum levels have been proved to be more sensitive than the CK levels to detect myocardial injury in many clinical situations including unstable angina pectoris. In clinically suspected myocarditis, the serum TnT level is elevated even in the absence of any histologic signs of myocarditis.[92,93] In autoimmune murine myocarditis, cardiac troponin is a more sensitive marker than creatine kinase MB isoform (CK-MB),[94] and an elevated troponin level clearly indicates myocarditis[93,94]. Another study shows that CPK-MB is significantly raised in 78.5% of cases.[81] Although serum Trop-T is specific for myocardial injury, it was positive in their 12 cases.

MATERIALS AND METHODS

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An observational cross sectional study was carried out among patients admitted in General Medicine ward, Govt. Stanley Medical College Hospital, Chennai. An informed consent was obtained from all patients who underwent the study. Both men and women more than 12 years of age and less than 65 years of age were included. All patients had a thorough history and physical examination and investigation as follows:

Name, Age, Sex, Duration of hospital stay, Duration of symptoms at admission. The patients were evaluated for symptoms of Fever, Myalgia, Calf tenderness, Arthralgia, Conjunctival suffusion, Headache, Retroorbital pain, Chills & rigors, Cough, Chest pain, Palpitation, Hemoptysis, Hematuria, Shock/ giddiness, Dyspnoea, Nausea & vomiting, Facial flushing, Rash, Bleeding tendency, Oliguria, Jaundice, Diarrhoea, Abdominal pain.

All patients were thoroughly examined for Consciousness, Orientation, Built/ nourishment, Pulse, Blood Pressure, Respiratory Rate Temperature, O2 saturation, Pallor, Icterus, Cyanosis, Pedal edema, JVP. System examination included evaluation of Apical impulse, Heart Sounds, Murmurs, Added sounds, Breath sounds, Hepatomegaly, Splenomegaly, Ascites, Higher function, meningeal signs.

General investigations included Hemoglobin, Total and differential count, ESR, platelets, PCV, Urine for sugar albumin and deposits, Random Blood Sugar, B. Urea, S. creatinine, S. Sodium, S. Potassium, S. Bilirubin S. ALT, S. AST S. Alkaline phosphatase, T. Protein, S. Albumin, Bleeding Time, Clotting time.

Fever profile included Peripheral smear, QBC for malarial parasite, MSAT for Leptospirosis, Dengue IgM, Blood Widal to rule out Enteric Fever and USG of Abdomen.

X-Ray Chest, ECG, Echocardiogram, S. Total CK and S. CK- MB was done in all patients as a part of evaluation of cardiac status. 12 Lead ECG was taken in all patients at the time of admission and at the day of discharge. All the parameters in ECG were estimated according

to standard guidelines. Repeat Echocardiogram was done in all patients whose initial Echo findings were abnormal.

Diagnosis of Malaria (see Annexure- 1) and Dengue (see Annexure- 2) were based on WHO guidelines. Leptospirosis was diagnosed based on Modified Faine's Criteria (see Annexure - 3). Patients who were found to have laboratory investigations or serology for more than one illness were considered to have co-infection. Serology to diagnose Dengue and Leptospirosis and Widal to rule out Enteric fever were done after 7 days of onset of illness.

Total CK (IU/L) was estimated by the NAC Activated Method. CKMB (IU/L) was estimated by immune-inhibition method. The normal values for Total CK and CKMB were as follows:

Total CK – 24-180 IU/L;

CKMB – 25 IU/L.

CKMB values were considered normal or insignificant in cases were the Total CK levels were less than 80 IU/L and if the CKMB levels were less than 50 IU/L. A CKMB value of more than twice the upper limit of normal range (50 IU/L) was considered as elevated in patients with Total CK levels in the range of 80-180 IU/L. In patients with Total CK more than 180 IU/L, percentage of CKMB activity (CKMB index) was calculated by the formula:

[CKMB (IU/L)/ Total CK (IU/L)] X 100.

An Index of more than 6% was considered to be indicative of myocardial injury in patients with Total CK more than 180 IU/L and CKMB more than 50 IU/L.

Patients who were found to be positive for other co-existing infectious disease during the work up were excluded from the study. Care was taken to see that patients were not having any illness or on any drugs which could alter the ECG or echo findings. The exclusion criteria for the study were as follows:

EXCLUSION CRITERIA

- Patients < 12 years and > 65 years of age, pregnancy.

- Any febrile illness which does not satisfy the criteria for the above 3 diseases.
- Patients diagnosed to have any other febrile illnesses during the course of the study.
- Patients with heart disease (congenital or acquired), Hypertension, Diabetes.
- Those with features of liver or renal failure or dyselectrolytemia.
- Patients with chronic pulmonary, renal, gastrointestinal or liver disease.
- Patients with neuromuscular disorders, seizure disorders.
- Patients with malignancies, hematological disorders or coagulopathies.
- Patients undergoing treatment for chronic diseases (eg- thyroid, rheumatoid arthritis, connective tissue disorders, tuberculosis etc).

All patients were treated according to the standard guidelines followed in the institution. Mild leptospirosis cases were treated with oral doxycycline. Severe leptospirosis was treated with I.V. penicillin (or) ceftriaxone.

All uncomplicated malaria patients were treated with Chloroquine (25mg/kg) with Doxycycline 100mg BD for 3 days. In case of no clinical response, patients were switched over to quinine 10mg/kg 8th hourly or Oral Artemisinin Combination Therapy (See Annexure). All complicated malaria patients showing organ dysfunction were started on quinine 10mg/kg 8th hourly oral or intravenously in dextrose solution or IV Artesunate 2.4 mg/kg/day for 5 days along with oral Doxycycline 100mg BD for 7 days. Primaquine was given for 15 days in those with Plasmodium vivax infection.

Dengue was treated symptomatically as per WHO guidelines (see Annexure…), with special attention being given to fluid resuscitation and monitoring for thrombocytopenia and hemoconcentration.

Patients were discharged when they were afebrile for 3 continuous days OR the relief of other associated symptoms, whichever came late. The data were tabulated and evaluated using *Microsoft Excel Worksheet 2010* and *SPSS 17* statistical analysis software.

RESULTS & OBSERVATIONS

RESULTS & OBSERVATIONS

A total of 100 patients were evaluated in this study. 56 of them were males and the rest 44 were females. The disease-wise split up of the 100 cases is as follows:

Table 4

Disease	No. of Cases	Percentage
Leptospirosis	35	32
Malaria	36	33
Dengue	39	35

Co-infection was found in 10 patients.

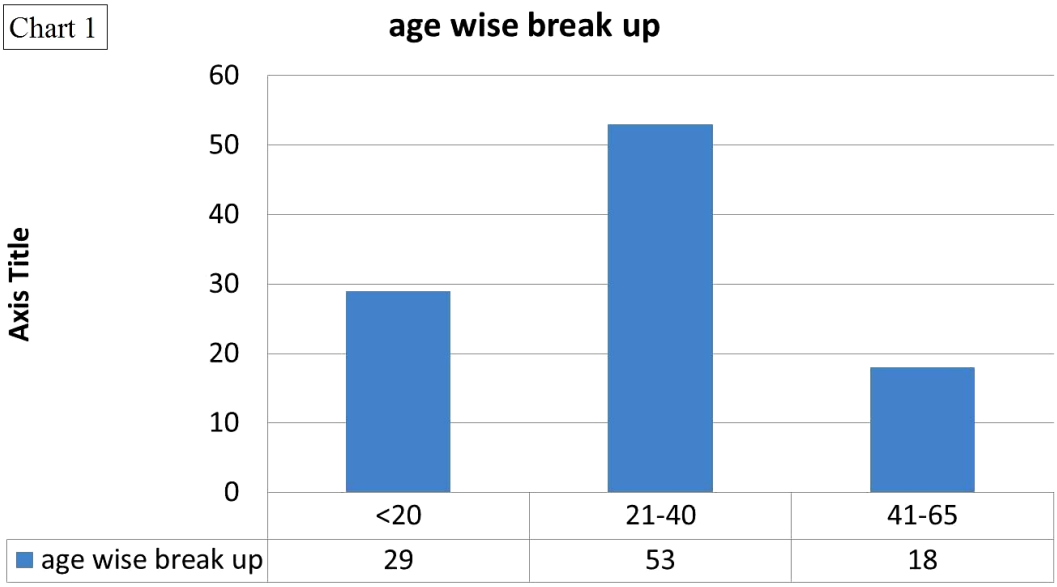
Table 5

Co-infection	No. of Cases
Leptospirosis + Malaria	2
Leptospirosis + Dengue	6
Malaria + Dengue	2

Of the 100 patients in the study 97 patients were discharged on recovery from illness. Two patients with severe leptospirosis and one patient with Dengue Shock Syndrome expired.

Age wise distribution of patients in the study is shown in the bar chart below.

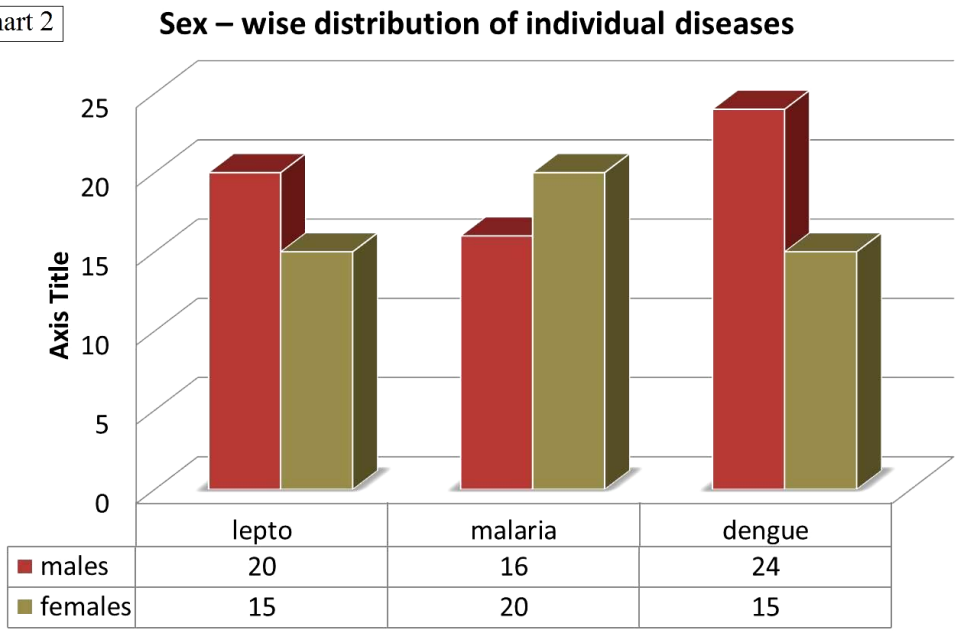
Chart 1



As can be seen, majority of patients belonged to the 20 -40 years age group.

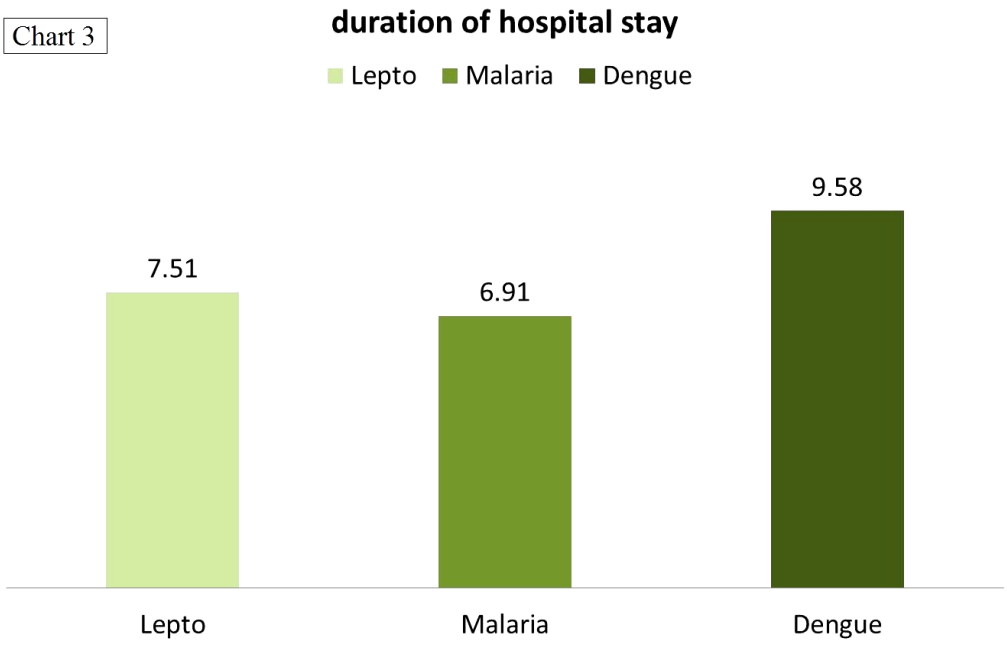
Sex – wise distribution of individual diseases is as shown in chart 2.

Chart 2



The average duration of hospital stay was slightly higher in dengue as shown in Chart 3

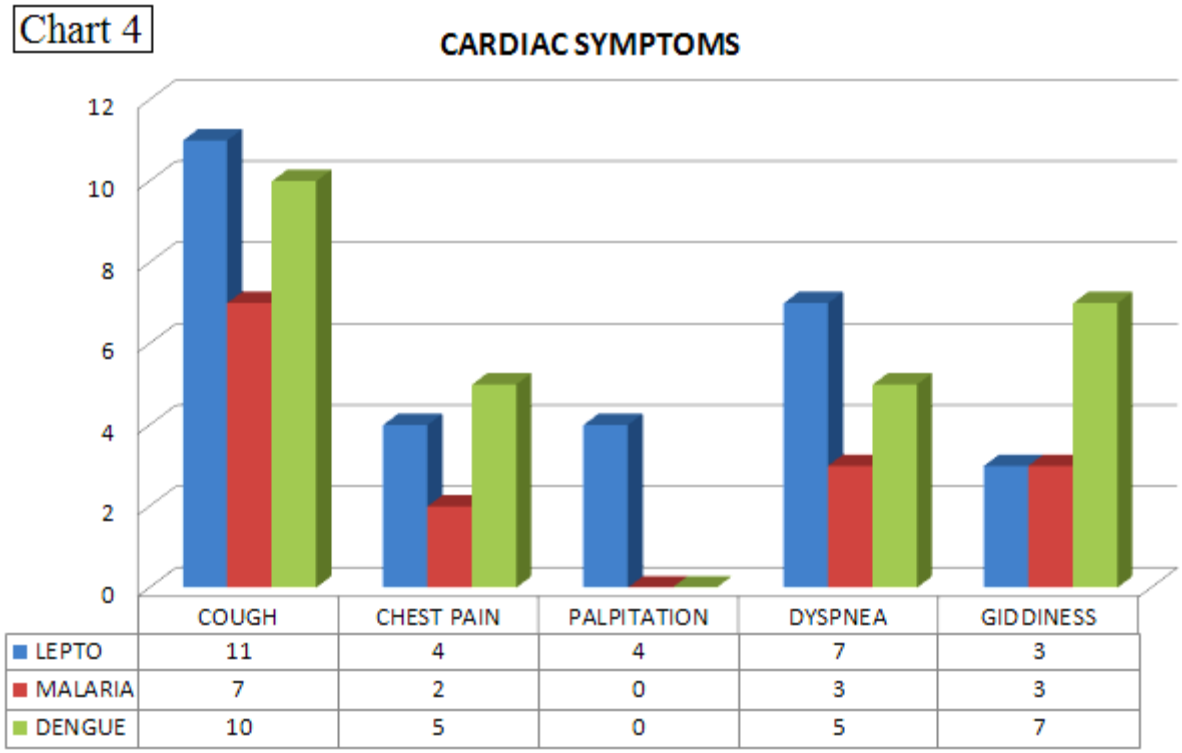
Chart 3



Cardiac symptoms

Analysis of cardiac symptoms showed that patients with leptospirosis and dengue had more cardiac symptoms compared to malaria. Also, it was noted that hypotensive symptoms were more common in dengue (Chart 4).

Chart 4



Pulse Rate

Table 6

	<60/min (%)	61-99 (%)	>100 (%)
Lepto (n=35)	**4 (12%)**	**12 (34%)**	**19 (54%)**
Malaria (n=36)	**0**	**9 (25%)**	**27 (75%)**
Dengue (n=39)	**8 (21%)**	**13 (33%)**	**18 (46%)**

As seen in Table 6, Comparison of pulse rate showed more number of patients with dengue having bradycardia. Tachycardia was more prevalent in malaria.

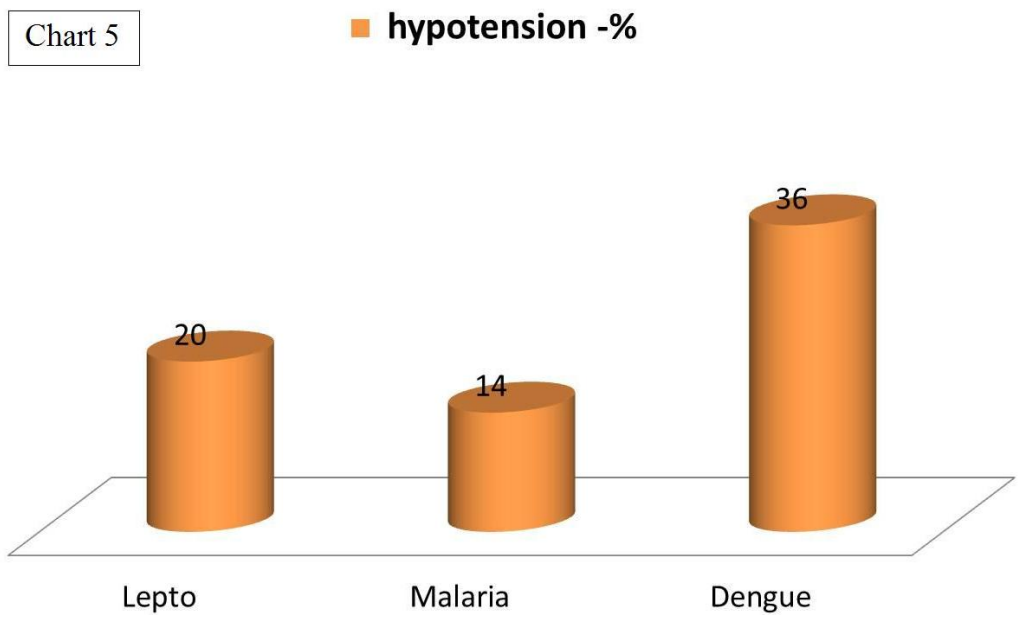
Systolic Blood Pressure

Table 7

	<90 mmHg (%)	91-100 mmHg (%)	100-120 mmHg	>120 mmHg
Lepto (n=35)	**7 (20%)**	**7 (20%)**	**20 (57%)**	**1 (3%)**
Malaria (n=36)	**5 (14%)**	**15 (42%)**	**12 (33%)**	**4 (11%)**
Dengue (n=39)	**13 (36%)**	**9 (25%)**	**13 (36%)**	**1 (3%)**

The above data in Table 7 shows that hypotension (SBP < 90 mmHg) is more common in Dengue (Chart 5).

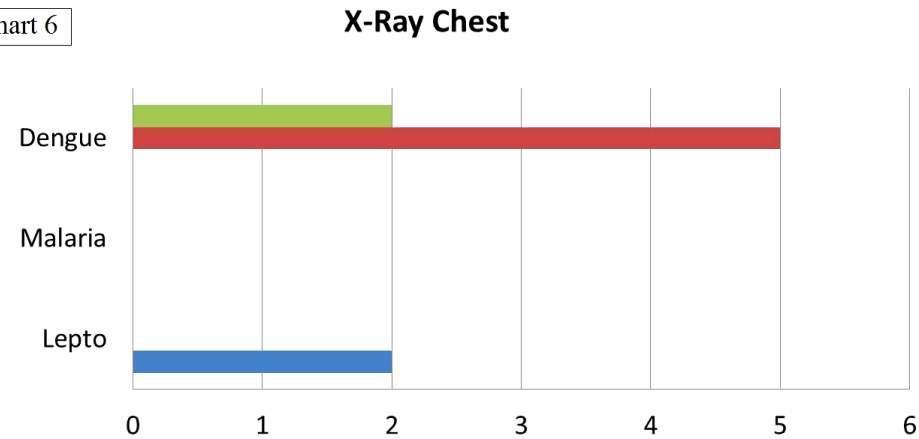
Chart 5



Chest X ray

Out of the 100 patients, only nine had abnormal chest X- ray findings. (chart 6).

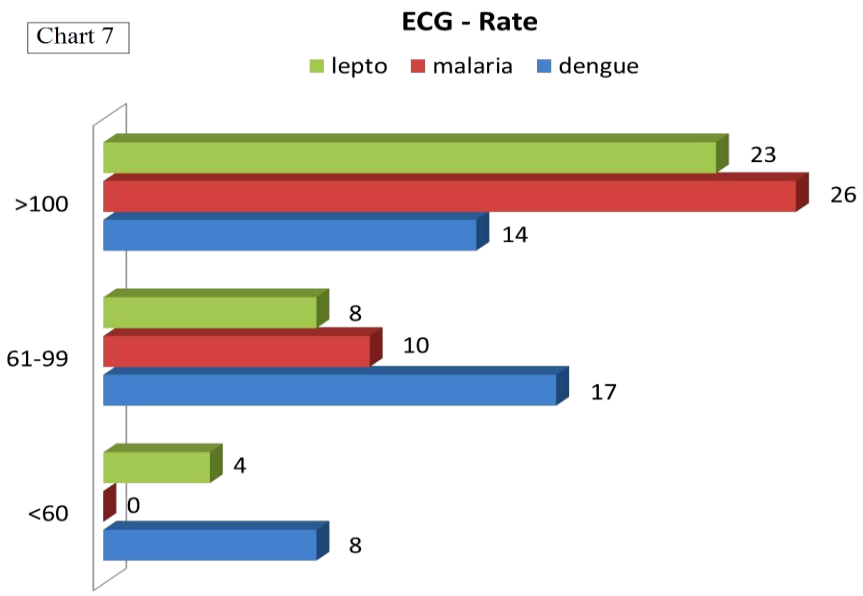
Chart 6



	Lepto	Malaria	Dengue
L pl. effusion	0	0	2
b/l pl.effusion	0	0	5
pul. Edema	2	0	0

ECG rate

The ECG rate corresponded to the pulse rate. Bradycardia was more commonly seen in dengue. They also had more number of people with normal heart rate during the febrile period. Interestingly, none of the 36 patients with malaria had bradycardia (Chart 7).



Rhythm Abnormalities

Rhythm abnormalities were rarely observed in the study. Transient Atrial ectopics were noted in two patients with Dengue and transient ventricular ectopics were noted in 1 patient with Leptospirosis. No abnormalities were observed in Malaria. All three irregularities were normalized in the repeat ECG

PR interval abnormalities

First degree heart block was observed in 4 patients with dengue and 2 patients with malaria. No abnormalities were detected in patients with leptospirosis (Table 8).

Table 8

	1st degree Heart Block (%)
Lepto (n=35)	**0**
Malaria (n=36)	**2 (5.55%)**
Dengue (n=39)	**4 (10.25%)**

QRS abnormalities & QT prolongation

Ventricular repolarization abnormalities were infrequent. The observations are as follows (Table 9). Low voltage complexes were seen in two patients with pericardial effusion. The conduction abnormalities normalized in the repeat ECG. None had QT prolongation.

Table 9				
	Incomplete RBBB	Low Voltage	LAFB	QT prolongation
Lepto (n=35)	**2 (5.71%)**	**0**	**1 (2.85%)**	**0**
Malaria (n=36)	**0**	**0**	**0**	**0**
Dengue (n=39)	**2 (5.12%)**	**2 (5.12%)**	**0**	**0**

ST – T Changes

ST – T changes were commonly observed in the 3 groups. Abnormal ST segment changes and T wave abnormalities were observed in 40 out of the 100 patients under study. The disease – wise split up is as in Chart 8 and Chart 9. The changes were least common in Malaria.

Chart 8

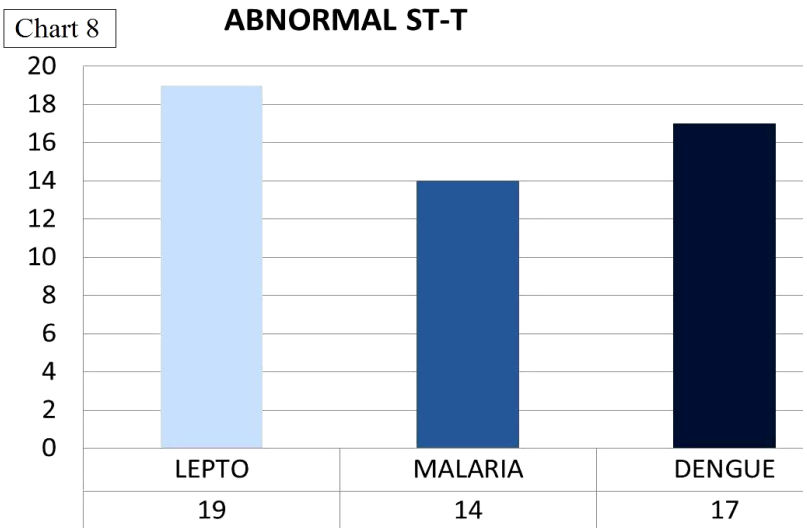
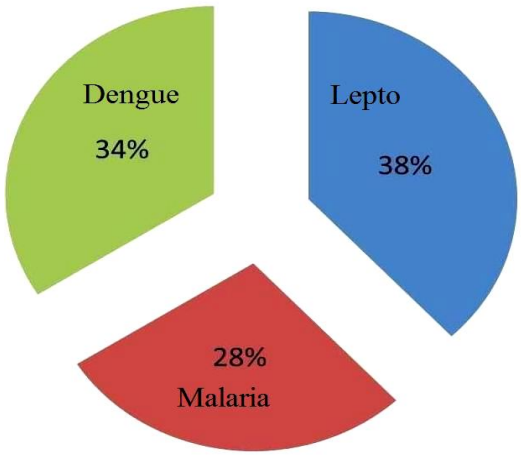
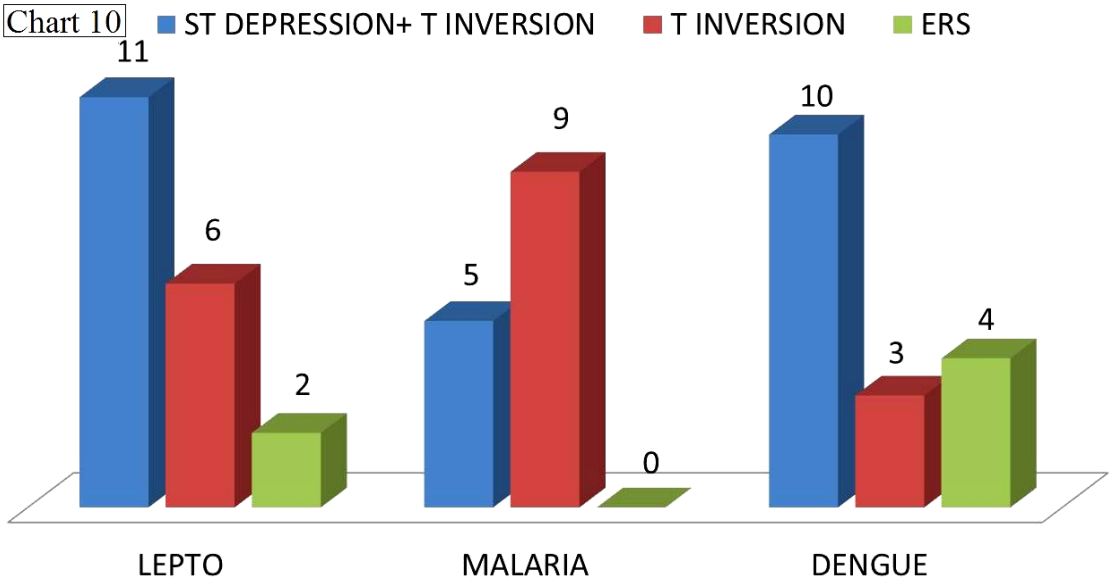


Chart 9



ST depression and T inversion together were more common in Leptospirosis and Dengue while isolated T inversion was more common in Malaria (Chart 10). None of the patients had ST segment elevation. Early repolarisation abnormalities were observed in few patients with Dengue and Leptospirosis.

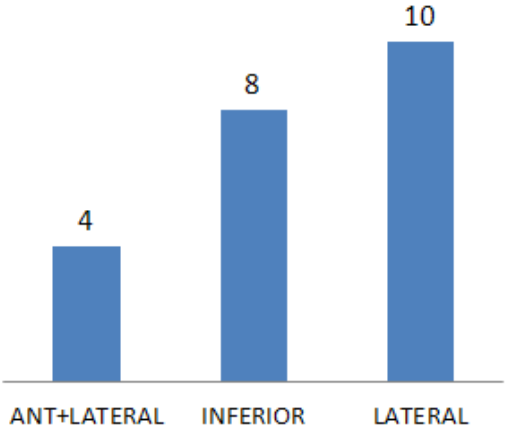
Chart 10



The ECG leads showing the ST – T changes were as shown in Charts 11, 12 and 13. Changes in I, aVL, V5, V6 was termed as lateral, II, III, aVF as inferior and changes in I, aVL, V1-V6 was termed as anterolateral. As seen below the ST – T changes were more frequent in the lateral leads in Leptospirosis. They were evenly distributed in Malaria. Dengue showed antero-lateral preponderance. More wide spread repolarization abnormalities were observed in Dengue.

Chart 11

LEPTO



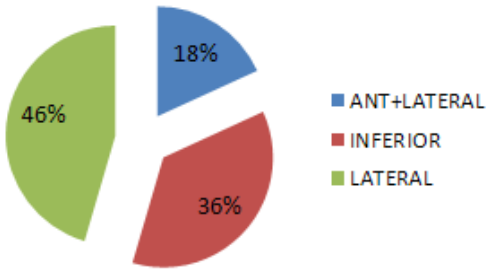
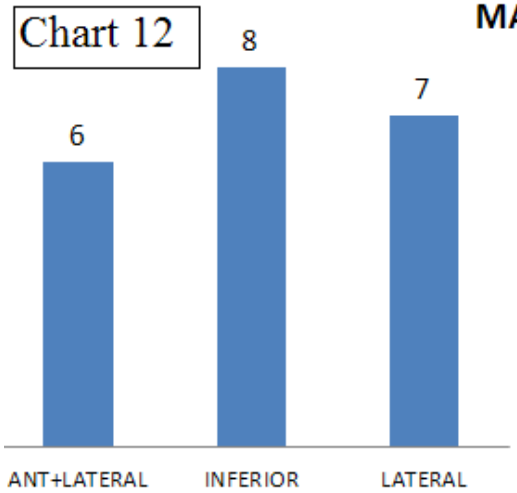


Chart 12

MALARIA



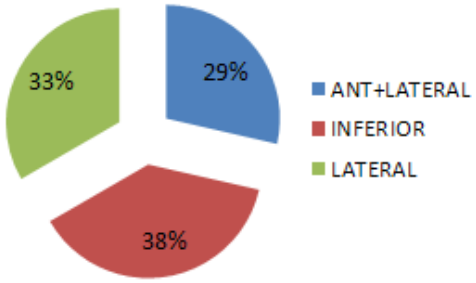
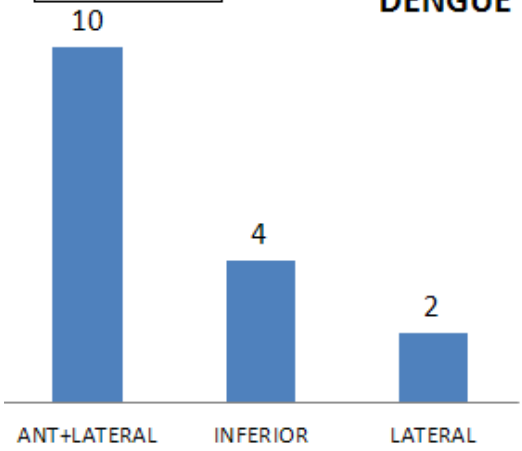
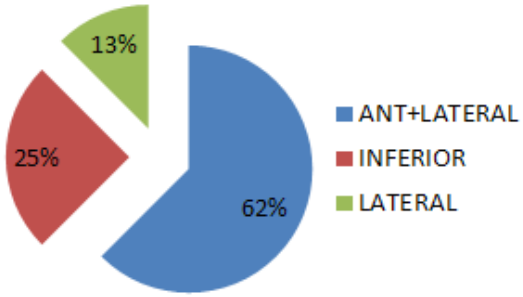


Chart 13

DENGUE





Total Creatine Kinase and Creatine Kinase – MB levels

The distribution of Total CK levels is as in Table 9

Table 9	Total CK		
	<80 IU/L	80 – 180 IU/L	> 80 IU/L
Lepto (n=35)	**2 (5.71%)**	**15 (42.85%)**	**18 (51.42%)**
Malaria (n=36)	**9 (25%)**	**16 (44.4%)**	**11 (30.55%)**
Dengue (n=39)	**4 (10.2%)**	**15 (38.46%)**	**20 (51.28%)**

Total CK elevation was much more commonly observed in Leptospirosis and Dengue, compared to Malaria.

The CKMB values and the CKMB relative index were distributed as below (Table 10).

Table 10	CKMB			
	Normal	> 50 IU/L	Index > 6	**HIGH (%)**
Lepto (n=35)	**16**	**4**	**15**	**19 (54.28%)**
Malaria (n=36)	**27**	**3**	**6**	**9 (25%)**
Dengue (n=39)	**22**	**3**	**14**	**17 (43.58%)**

As can be seen from the above table, the CKMB levels were significantly elevated in Leptospirosis and Dengue, compared to Malaria. This correlates well with the ST- T changes observed in ECG.

CKMB and ST-T changes in ECG

The relation between CKMB and ST-T changes in ECG is described below (Table 11).

Table 11	**NORMAL ST-T**	**ABNL ST-T**
Normal CKMB		
Lepto (n=16)	**11 (68.75%)**	**5 (31.25%)**
Malaria (n= 27)	**18 (66.67%)**	**9 (33.33%)**
Dengue (n=22)	**15 (68.18%)**	**7 (31.82%)**
High CKMB		
Lepto (n=19)	**8 (42.11%)**	**11 (57.89%)**
Malaria (n=9)	**4 (44.44%)**	**5 (55.55%)**
Dengue (n=17)	**7 (41.17%)**	**10 (58.82%)**

As seen above, the CKMB values correlated well with the ST-T changes in ECG. This was more conspicuously observed in patients with Leptospirosis and Dengue.

Echocardiogram

The Echo was normal in 68 of the 100 patients under study. 32 patients had abnormal echo findings. The disease – wise break up is as in Table 12.

Table 12

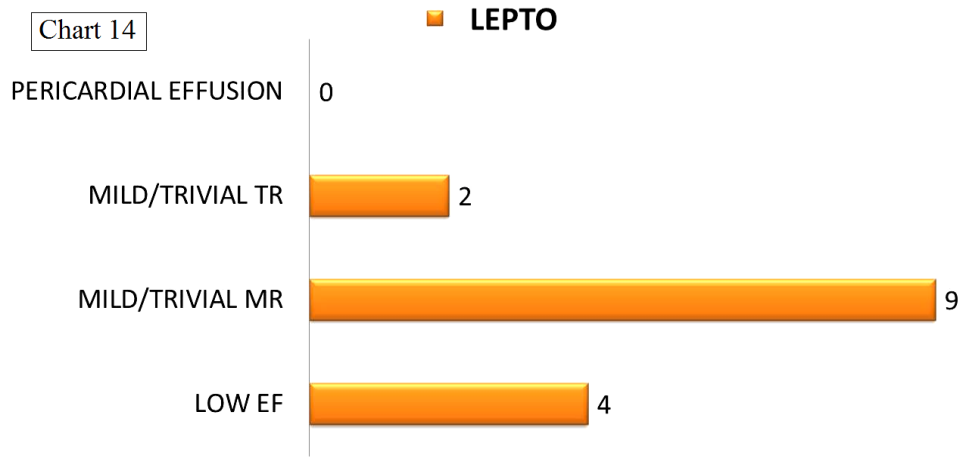
	Normal Echo	Low Voltage
Lepto (n=35)	**22 (62.85 %)**	**13 (37.14 %)**
Malaria (n=36)	**31(86.11 %)**	**5 (13.88 %)**
Dengue (n=39)	**20 (51.28 %)**	**19 (48.71 %)**

More than one third of patients with Leptospirosis had abnormal echo findings whereas almost half of the patients with Dengue had abnormal echo findings. Echocardiographic changes were observed less commonly in Malaria.

Echocardiogram in Leptospirosis

The various manifestations in Leptospirosis is as shown in Chart 14:

Chart 14

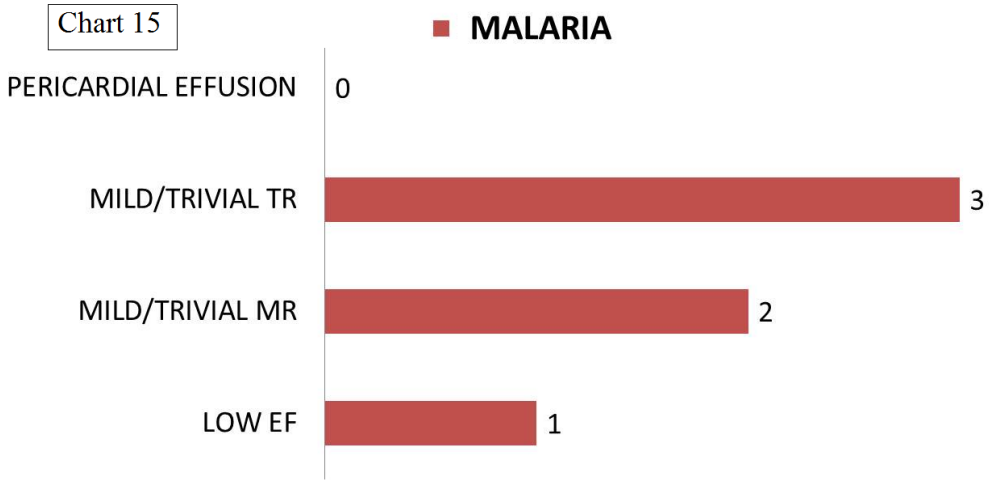


Mild or trivial valvular regurgitation was the most common echo finding in Leptospirosis. Low Ejection fraction was observed in 4 cases. Two of the patients with low ejection fraction expired.

Echocardiogram in Malaria

Only 5 patients with Malaria had abnormal echo findings (Chart 15).

Chart 15

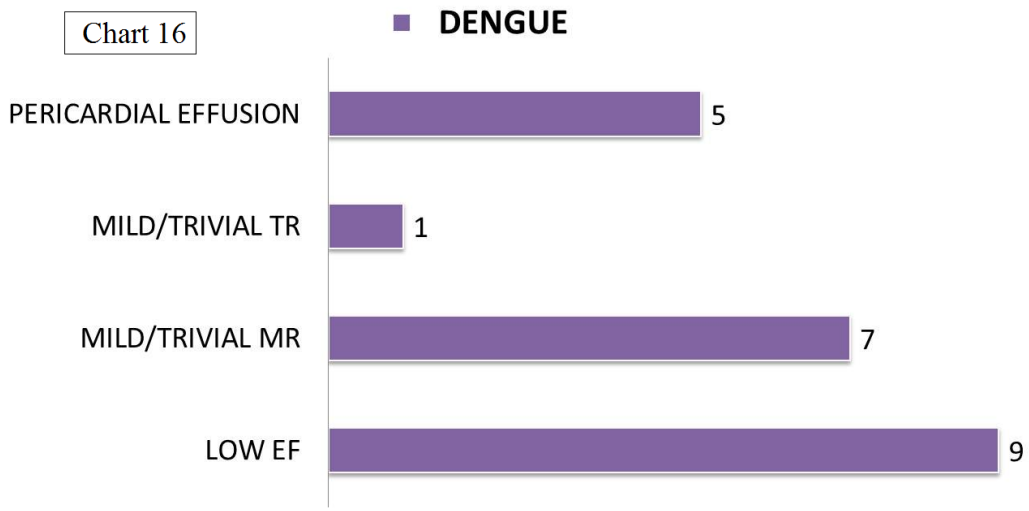


Mild valvular regurgitation was the commonest finding in Malaria. 1 patient had mildly depressed ejection fraction.

Echocardiogram in Dengue

The following findings were observed in Dengue (Chart 16)

Chart 16



Valvular regurgitation was observed in 8 patients with Dengue. But the most common finding was a mildly depressed ejection fraction. 5 patients had mild pericardial effusion. The lowest recorded Ejection Fraction was 40%. Three patients with depressed ejection fraction had mild pericardial effusion.

Heart Rate and Depressed Ejection Fraction in Dengue

The comparison of heart rate in dengue patients with depressed ejection fraction is shown in Table 13:

Table 13	
Depressed ejection fraction n= 9	
Bradycardia	Tachycardia
7	**2**

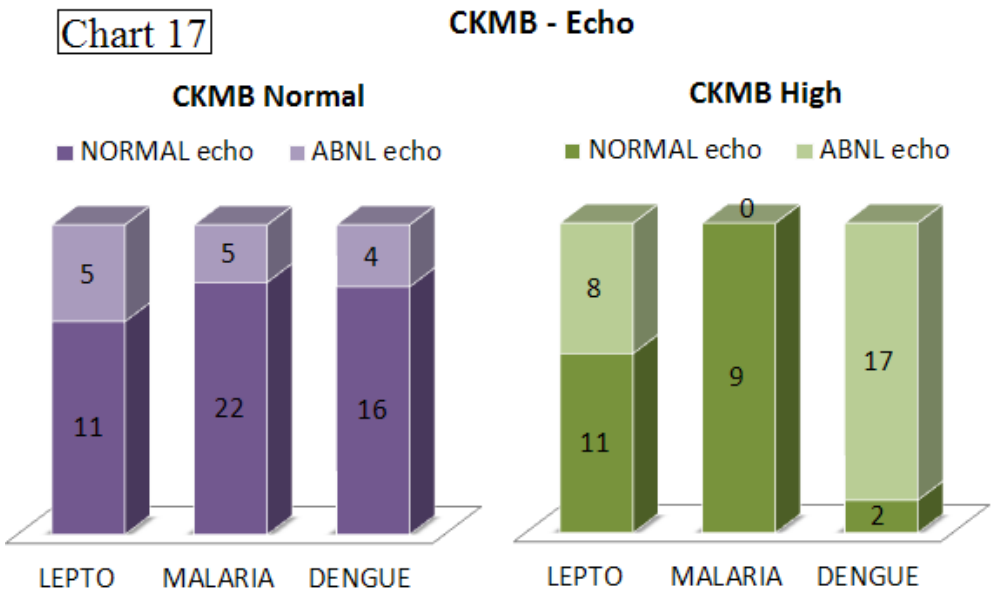
Seven out of the nine patients with depressed ejection fraction had bradycardia. The remaining two patients had tachycardia.

CKMB and Echocardiogram findings

The relation between CKMB and Echo findings is ECG is explained in Table 14 and Chart 17

Table 14	Normal CKMB	High CKMB
Normal ECHO		
Lepto (n=22)	**11 (50%)**	**11 (50%)**
Malaria (n=31)	**22 (70.96%)**	**9 (29.03%)**
Dengue (n=20)	**18 (90%)**	**2 (10%)**
Abnormal ECHO		
Lepto (n=13)	**5 (38.46%)**	**8 (61.53%)**
Malaria (n=5)	**5 (100%)**	**0**
Dengue (n=19)	**2 (10.53%)**	**17 (89.47%)**

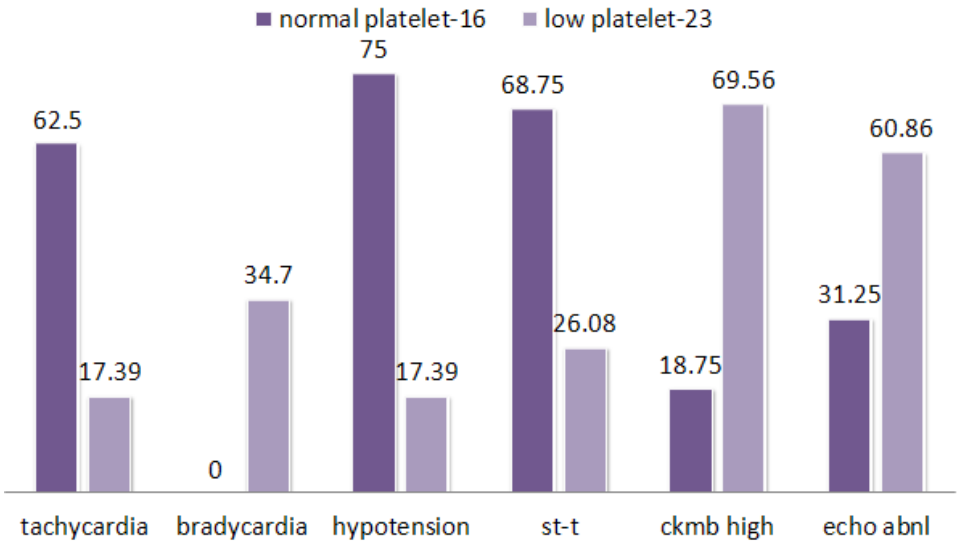
Chart 17



The statistics above show that higher CKMB values correlated with echocardiographic abnormalities. Out of 19 patients with Dengue who had an abnormal echo finding, 17 had elevated CKMB. Interestingly the five Malaria patients with abnormal echo findings had normal CKMB levels.

Thrombocytopenia and Cardiovascular Manifestations in Dengue

Chart 18 **Thrombocytopenia and CVS in Dengue**



As seen in Chart 18, incidence of bradycardia, high CKMB values and echo abnormalities were more in patients with thrombocytopenia. Patients with normal platelet count had higher incidence of tachycardia, hypotension and ST-T changes.

Sex- wise distribution of Cardiovascular Findings in Leptospirosis

The observations in Table 15 show that the cardiovascular manifestations of leptospirosis were more prevalent in males. Hypotension was more commonly observed in females

Table 15	Males	Females
Tachycardia	10	12
Bradycardia	4	0
Hypotension	2	5
ST-T	10	9
CKMB High	13	6
Echo Abnormal	8	5
TOTAL	**49**	**37**

Sex- wise distribution of Cardiovascular Findings in Malaria

Table 16	Males	Females
Tachycardia	16	20
Bradycardia	0	0
Hypotension	2	3
ST-T	2	12
CKMB High	4	5
Echo Abnormal	3	2
TOTAL	**27**	**42**

Table 16 shows that the cardiovascular manifestations were more common in females with Malaria.

Sex- wise distribution of Cardiovascular Findings in Dengue

Table 17	Males	Females
Tachycardia	7	7
Bradycardia	8	0
Hypotension	8	8
ST-T	8	9
CKMB High	13	6
Echo Abnormal	13	6
TOTAL	**57**	**36**

Cardiovascular manifestations of Dengue were more common in males (Table 17). Bradycardia was seen only in males.

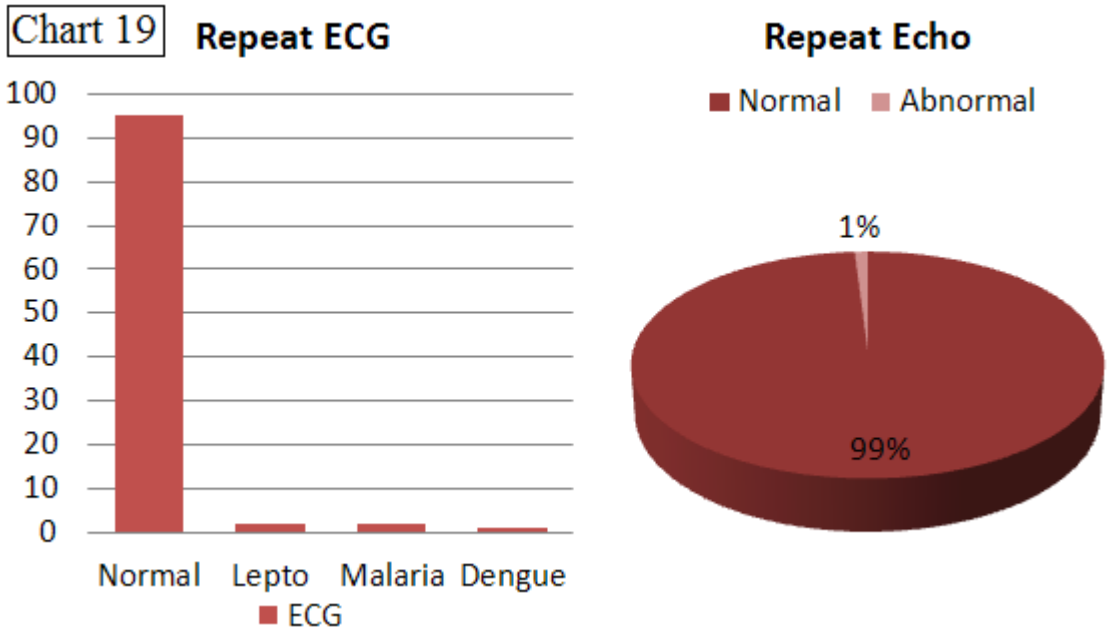
Comparing both sexes in all three infections, it was observed that tachycardia and hypotension was more common in females while males are more prone to develop bradycardia, CKMB elevations and echocardiographic abnormalities.

Cardiovascular manifestations in co infections

Table 18	Lepto + Dengue (n=6)	Dengue + Malaria (n=2)	Lepto + Malaria (n=2)
Bradycardia	**1**	**0**	**0**
Tachycardia	**4**	**1**	**2**
Hypotension	**3**	**1**	**0**
High CKMB	**2**	**0**	**2**
ST-T Changes	**5**	**2**	**1**
Echo Abnormal	**5**	**0**	**0**
Death	**0**	**0**	**0**

Table 18 shows the cardiovascular manifestations in co infections. Co-infection was seen in 10 patients (see table 5). The existence of co infection does not seem to increase the morbidity in this study group. Tachycardia and ST-T changes were common in all groups while bradycardia was seen only in one patient. Valvular regurgitation was the only finding observed in 5 abnormal echocardiograms.

Repeat ECG and Echocardiogram at discharge.



As seen in Chart 19, the repeat electrocardiogram was normal in only 95 patients. Of the 5 ECGs which showed persistant ECG changes, 3 were of patients who died.

Repeat Echo was normal in 99 %. The only abnormal second Echo observed was in a patient who expired.

Mortality

Two persons with severe leptospirosis and one patient with Dengue Shock syndrome died during the study.

DISCUSSION

DISCUSSION

The cardiovascular manifestations of three commonly occurring infectious diseases of North Chennai – namely Leptospirosis, Malaria and Dengue, were evaluated in this observational study. The patients who were included in the study had no previous or current cardiac illness. Neither were they on any medications which could probably cause ECG alterations. All patients found to have other co infections like pneumonia, Urinary Tract Infections, Enteric Fever were excluded from the study. Patients who were diagnosed to have the diseases under study but suffered other metabolic or biochemical complications like hypoglycemia, Diabetic ketoacidosis, dyselectrolytemia, renal failure etc. were also excluded.

Males made up 56 % of the study population, while 44 % were females. Diagnosis of Leptospirosis was made in 35 patients, Malaria in 36 patients and 39 patients in the study had Dengue (Table 4). Co infection was seen more commonly with Leptospirosis and Dengue (Table 5).

Of the 100 patients in the study 97 patients were discharged on recovery from illness. Two patients with severe leptospirosis and one patient with Dengue Shock Syndrome expired.

Age and Sex

Majority of patients in the study belonged to the age group of 20 – 40 years (Chart 1). Even though these diseases affect all age groups equally, many patients above 40 years had to be excluded from the study because of other comorbidities like Diabetes, CAD, Hypertension etc.

Males and females affected were almost equal in number in Malaria and leptospirosis. In case of Dengue, males were affected much more in number (Chart 2). This goes hand in hand with three independent studies from epidemics in India and Singapore found nearly twice the number of male patients compared to females (Lucknow and Singapore both report male to female ratios of 1.9:1 and Delhi 1:0.57).[63,64] It is widely recognised that in many of the Asian

communities, lower disease incidence in women may be a statistical artefact related to lower reporting and care-seeking for women from traditional practitioners who do not report to public surveillance systems. Determining sex differences, both in infection and severity of disease, requires well-designed and targeted studies to capture both biological and social factors that drive disease patterns in a community.

Duration of Hospital Stay

The average duration of hospital stay was slightly higher in Dengue (9.58 days) compared to Leptospirosis and Malaria (7.51 and 6.91 days respectively) as seen in Chart 3. This was mainly because the bradycardia and LV systolic dysfunction in patients with Dengue took longer time to normalize.

Cardiovascular symptoms.

Cough which was mostly dry or with mild mucoid expectoration was a common symptom in 28 patients (Chart 4). Dyspnea mostly NYHA class II was the second most common cardiac symptom. It was more common in Leptospirosis and Dengue (7 and 5 cases) compared to Giddiness or tiredness and fatigability was the next common symptom, seen in 13 patients. This was more observed in Dengue (7 versus 3 cases each in malaria and Leptospirosis. Chest pain, mostly atypical was a symptom in 10 patients. Palpitation was seen least and all 4 cases were Leptospirosis.

Apart from the common complaints of fever, headache, myalgia, rigor, it was found during the study that cardiac symptoms were present in many patients. Cardiovascular symptoms were more common in Leptospirosis and Dengue and less common in Malaria. The symptoms were mild in most of the cases and were overshadowed by the fever and myalgia which was uniformly present in the three groups. Careful questioning based on the proforma brought out the symptoms in these patients. Florid symptoms of NYHA Class IV dyspnea and chest pain were present in 2 patients who died due to severe leptospirosis. Giddiness and

fatigability in Dengue patients can be attributed to the presence of bradycardia, hypotension and depressed ejection fraction noted in many patients (see below). Dehydration and hypovolemia along with depletion of intravascular fluid due to capillary leak syndrome could be additional factors leading to increased incidence of these symptoms.

Pulse rate

Sinus tachycardia was the most common rate disturbance encountered in the study (Table 6), seen in 54 % of patients with Leptospirosis, 75 % of Malaria and 46 % of Dengue In the background of fever this was the most common abnormality expected. Sinus bradycardia was seen in 21% of patients in dengue and 12% patients with Leptospirosis. No patient with Malaria had bradycardia. The ECG findings were similar to the physical findings.

Bradycardia has been reported in few cases with Leptospirosis.[65,66] There are many studies quoting the high incidence of bradycardia in Dengue.[67,68,69] The current study also supports the view that bradycardia is a common manifestation in Dengue.

Blood pressure

The data showed that hypotension (SBP<90 mmHg) was more common in Dengue (36%) compared to Leptospirosis (20%) and Malaria (14%) (Table 7, Chart 5). The direct cardio-suppression by Dengue virus is a possibility for this pattern. Dengue viral antigen, associating with a myocardial receptor site, thereby triggering off an immunological response is also another possibility in susceptible individuals.[59]

Chest X ray

Mild Pleural effusion was observed in 7 patients with Dengue (Chart 6). Two patients with severe Leptospirosis who died had X- ray findings suggesting acute pulmonary edema. Cardiogenic pulmonary edema occurs more frequently than does ARDS in patients with leptospirosis. The severity of this may range from an asymptomatic radiographic finding to overt cardiogenic shock with acute pulmonary edema. Several studies report radiographic

evidence of cardiogenic pulmonary edema, but it must be remembered that this is difficult to distinguish from ARDS or hemorrhagic pneumonitis by chest radiography alone.[70,71,72] Hemodynamic data or echocardiographic evidence of left ventricular dysfunction is required to adequately distinguish between these disorders.

Rhythm Abnormalities in ECG

Rhythm abnormalities were relatively infrequent in the population under study. Transient Atrial ectopics were noted in two patients with Dengue and transient ventricular ectopics were noted in 1 patient with Leptospirosis. No abnormalities were observed in Malaria.

First degree heart block was observed in 4 patients with Dengue and 2 patients with Malaria. No abnormalities were detected in patients with Leptospirosis. Ventricular repolarization abnormalities were infrequent. Low voltage complexes were seen in two patients with pericardial effusion. None had QT prolongation.

Rhythm disturbances also appear commonly in patients with leptospirosis. Atrial arrhythmias, including both atrial fibrillation and atrial flutter, are frequently encountered.[70,71] Premature ventricular contractions are common, and one case of ventricular tachycardia has been reported.[71] In addition, both first- and second-degree heart block and junctional rhythms have been described in patients with leptospirosis. As with the ECG abnormalities, rhythm disturbances associated with leptospirosis usually resolve early in the course of the illness.[73]

In a Brazilian study of 157 cases, atrial fibrillation was the most frequent (10.8%) while alterations in ventricular repolarization (38.9%) and first-degree atrioventricular block (10.2%) were other frequent findings.[74] Complete heart block has been reported in a fatal case of falciparum malaria.[39] Electrocardiographic abnormalities have been reported in 44-75% of patients with viral hemorrhagic fever, and prolongation of the PR interval or sinus bradycardia commonly occurs. Some have reported atrio-ventricular block in variable degrees. This may represent a transient functional impairment which may resulting from

abnormalities in autonomic tone, or localized pathology, such as minute bleeding in the area of the SA node.[75,76]

The rhythm abnormalities were uncommon and non life threatening in all three diseases in this study. This could be due to the presence of serovars or serotypes which cause mild illness being more prevalent in this area. Another reason could be the early diagnosis and treatment of these infectious diseases. All the abnormalities observed were completely reversed at discharge.

QRS changes and QT prolongation

These changes were uncommon. Intraventricular conduction blocks in the form of incomplete RBBB were present in two patients with Leptospirosis and Dengue. One patient with Leptospirosis had LAFB. Low voltage complexes were observed in two patents with Dengue who had mild pericardial effusion.

Delayed intraventricular conduction or bundle branch blocks have been reported in at least one review on leptospirosis but appear to be an infrequent finding.[77] The same hold true with Malaria and Dengue

ST – T Changes

ST – T changes were commonly observed in the 3 groups. Abnormal ST segment changes and T wave abnormalities were observed in 40 out of the 100 patients under study. ST – T changes occurred in 38% of Leptospirosis, 34% of Dengue and 28% of Malaria. None of the patients had ST elevation (Chart 8 & 9).

ST depression and T inversion together were seen in Leptospirosis (11/19) and Dengue (10/19) were as isolated T inversion was more common in malaria (9/14) (Chart 10).

ST – T changes were more commonly observed in the infero- lateral leads in Leptospirosis (82%), whereas anterolateral preponderance (75%) was noted in Dengue (Charts 11-13).

Both intraventricular conduction delay and nonspecific T wave changes were reported in the study by Sodeman and Killough of 80 cases of Weil's disease.[77] Of their eight patients who had clinical evidence of myocardial involvement, seven had either flattened or inverted T waves on an ECG, most commonly in the anterior or lateral leads. Another study of the ECG abnormalities of leptospirosis noted pronounced anterior ST-segment elevation in four patients.[73] In none of the patients in either study did clinical or laboratory evidence of myocardial infarction develop, and in cases where follow-up information was available, the ECG returned to normal after proper antibiotic therapy. In the latter series the authors postulated a localized myocarditis or pericarditis to explain these ECG findings.[73]

ST-T wave changes characteristic of pericarditis have been found in at least one patient with a leptospirosis-induced pericardial friction rub.[78] Even children have been found to have nonspecific ECG abnormalities in association with leptospirosis.[79]

ST – T wave changes are common in ECG in patients with Leptospirosis and Dengue and are less prevalent in Malaria. This study showed an inferolateral preference in Leptospirosis and anterolateral preference in Dengue. The explanation for these findings need further studies on cardiac imaging and perfusion. All changes reversed with treatment.

Total Creatine Kinase and Creatine Kinase – MB levels

Patients with Leptospirosis (51.42%) and Dengue (51.28%) had higher number of elevated Total CK levels as compared to Malaria (30.55%) (Table 9). This explains the involvement of muscle – both cardiac and skeletal – in these two disorders.

Elevated CKMB values where seen in Leptospirosis (54.28%) and Dengue (43.58%) compared to Malaria (25%) suggesting Leptospirosis and Dengue myocarditis (Table 10).

Normal CKMB levels were associated with lesser number of ST-T changes in all three groups. High CKMB values were associated with greater number of ST-T changes in all three groups, more common in Leptospirosis and Dengue.

Biomarkers of cardiac injury are elevated in a minority of patients with acute myocarditis but may help confirm the diagnosis. Western reports have shown that CK or its isoform (CK-MB) is not generally useful for noninvasive screening for myocarditis because of its low predictive value.[80] A study from New Delhi by Gupta et.al. showed that CKMB was also raised by a significant level in patients with dengue, thereby suggesting that it too can be useful as a marker for myocarditis.[81]

Echocardiogram

An abnormal echo was obtained in 37.14 % patients with Leptospirosis, 13.88 % of patients with malaria and 48.71 % patients with Dengue (Table 12).

Mild or trivial valvular regurgitation was seen in 11 cases with Leptospirosis, 9 of them being mitral. Four had low ejection fraction (Chart 14). Mild or trivial valvular regurgitation was seen in 5 patients with Malaria. One patient had mildly depressed EF (Chart 15). Valvular regurgitation was observed in 8 patients with Dengue. The most common finding was a depressed ejection fraction in 9 patients. Five patients had mild pericardial effusion. The lowest recorded Ejection Fraction was 40%. Three patients with depressed ejection fraction had mild pericardial effusion (Chart 15).

Of the 9 cases of Dengue with depressed ejection fraction, 7 had bradycardia and the rest two had tachycardia (Table 13). This implies a common pathogenesis with direct myocardial involvement by the virus being responsible.

61.53% of Leptospirosis with abnormal Echo had high CKMB. 89.47% of Dengue with abnormal Echo had high CKMB. CKMB was normal in the five Malaria patients with abnormal Echo. This shows that CKMB is a highly sensitive marker of Dengue myocarditis in this population. It is also useful in Leptospirosis. Its use remains questionable in Malaria. It is more indicative of LV dysfunction than for valvular regurgitation. All the echocardiographic abnormalities normalized with treatment.

The usefulness of Echo in diagnosis of myocarditis is well established. The common findings are LV systolic dysfunction with depressed ejection fraction, LV dilatation, diastolic dysfunction, valvular regurgitation and regional wall motion abnormalities suggesting myocarditis.[82]

RL Satarasinghe et.al. reported transient chamber dilatation with a good ejection fraction and jerky wall motion were the major abnormalities, which dominantly involved the right ventricle and a minor degree of atrioventricular valvular regurgitation.[59] The right ventricle (RV) showed dilation with associated tricuspid regurgitation in 57% (35/61) of patients, left ventricular dilation was observed less often in 21% (13/61) of patients, followed by duel chamber dilatation in 16% (10/61) of patients and isolated tricuspid regurgitation in 6% of patients. All had a satisfactory ejection fraction. CPK-MB values were not helpful in diagnosing myocardial involvement.[59] Wali *et al,* concluded that approximately 20% of those who developed DHF have a LV ejection fraction of less than 50%, and are likely to return to normal within a few weeks.[58]

In the New Delhi study, 4 cases (14%) had bradycardia in electrocardiography, and 4 cases (14%) had grade 1 diastolic dysfunction in 2D-echocardiography which is a manifestation of cardiac involvement. In other cases Echo was normal, but cardiac enzymes were significantly raised in most of these cases (serum CPKMB level and S. trop. T).[81]

Serum CKMB levels seemed to correlate well with Echo findings in this study, especially in Dengue. Sinus bradycardia also was associated with Echo abnormalities in Dengue in this study as opposed to some previous reports.[59] The heart seems to be relatively spared by Malaria in this study. The transient mild valvular regurgitation may represent mild valvulitis in these patients.

Thrombocytopenia and Cardiovascular Manifestations in Dengue

The occurrence of bradycardia, elevated CKMB values and echo abnormalities in Dengue were more common in patients with thrombocytopenia. Patients with normal platelet count had higher incidence of tachycardia, hypotension and ST-T changes (Chart 18). This suggests that thrombocytopenia along with bradycardia and elevated CKMB values are useful markers for predicting myocarditis in Dengue.

Sex- wise distribution of Cardiovascular Findings

Bradycardia, CKMB elevations and Echo abnormalities were common in Males with Leptospirosis. Hypotension was more common in females (Table 15). Tachycardia and ST-T changes were common in females with Malaria. Other parameters had equal sex distribution (Table 16). Males with Dengue had higher occurrence of Bradycardia, CKMB elevations and Echo abnormalities (Table 17).

Comparing both sexes in all three infections, it was observed that tachycardia and hypotension was more common in females while males are more prone to develop bradycardia, CKMB elevations and echocardiographic abnormalities.

Cardiovascular manifestations in co infections

The existence of co infection does not seem to increase the morbidity in this study group. Tachycardia and ST-T changes were common in all groups while bradycardia was seen only in one patient. Valvular regurgitation was the only finding observed in 5 abnormal echocardiograms (Table 18).

Repeat ECG and Echocardiogram at discharge.

The repeat ECG was normal in only 95 patients. Of the 5 ECGs which showed persistant ECG changes, 3 were of patients who died. Repeat Echo was normal in 99 %. The only abnormal second Echo observed was in a patient who expired (Chart 19).

Mortality

Two persons with severe Leptospirosis and one patient with Dengue Shock syndrome died during the study. Both patients who died of Leptospirosis presented late to the hospital, more than 2 weeks after onset of fever. Both had overt signs of heart failure and pulmonary edema, which was refractory to standard treatment. Both patients expired on the 3rd day in hospital. The Total CK and CKMB were grossly elevated indicating the widespread muscle damage, both skeletal and cardiac.

One patient with DHF/DSS presented after 6 days of fever with anasarca and features of cardiac decompensation. Persistant hypotension was present which was resistant to IV fluids, platelet transfusions and steroids. The patient died on day 9 of admission. Echo showed global LV systolic dysfunction with low ejection fraction, mild pericardial effusion. ECG showed persistant sinus bradycardia.

SUMMARY

SUMMARY

- In this study, a total of 100 cases were analysed. 35 had Leptospirosis, 36 had Malaria and 39 had Dengue. Lepto – Dengue co-infection was found in 6 patients
- Majority of patients in the study belonged to the age group of 20 – 40 years. Dengue was more common in males.
- Patients with Dengue had slightly prolonged duration of hospital stay (9.58 days) compared to Leptospirosis (7.51 days) and Malaria (6.91 days).
- Mild Non productive cough and NYHA class II dyspnea were the most common cardiovascular symptoms. Giddiness and fatigability were more seen in Dengue.
- Sinus tachycardia was common in Leptospirosis (54%), Malaria (75 %) and Dengue (46 %). Sinus bradycardia was seen in 21% of patients in dengue and 12% patients with Leptospirosis.
- 36% of Dengue had hypotension compared to 20% in Lepto and 14% in Malaria.
- Minimal Pleural effusion was the commonest radiographic finding in Dengue. Two patients with severe Leptospirosis had features of pulmonary edema in X-ray.
- Rhythm abnormalities and conduction blocks were rarely encountered in the study. 4 patients with Dengue had 1st degree heart block.
- Transient Incomplete RBBB was noted in two patients with Lepto and Dengue.
- ST-T changes occuredin 40% of the patients in total - 38% of Leptospirosis, 34% of Dengue and 28% of Malaria. ST depression and T inversion together were seen in Leptospirosis (11/19) and Dengue (10/19) were as isolated T inversion was more common in malaria (9/14).
- ST – T changes were more commonly observed in the inferior and lateral leads in Leptospirosis (82%), whereas anterior and lateral preponderance (75%) was noted in Dengue (Charts 11-13).

- Patients with Leptospirosis (51.42%) and Dengue (51.28%) had higher number of elevated Total CK levels as compared to Malaria (30.55%).
- Significantly elevated CKMB values where seen in Leptospirosis (54.28%) and Dengue (43.58%) compared to Malaria (25%) suggesting Leptospirosis and Dengue myocarditis.
- High CKMB values were associated with greater number of ST-T changes in all three groups, more common in Leptospirosis and Dengue.
- An abnormal echo was obtained in 37.14 % patients with Leptospirosis, 13.88 % of patients with malaria and 48.71 % patients with Dengue
- Mild or trivial valvular regurgitation was seen in 11 cases with Leptospirosis, 9 of them being mitral. Four had low ejection fraction.
- Depressed EF was present in 9 patients with dengue whereas 8 had Mild or trivial valvular regurgitation. 5 patients had mild pericardial effusion.
- Of the 9 cases of Dengue with depressed ejection fraction, 7 had bradycardia.
- Bradycardia, elevated CKMB values and echo abnormalities in Dengue were more common in patients with thrombocytopenia.
- Bradycardia, CKMB elevations and Echo abnormalities were common in Males with Leptospirosis. Hypotension was more common in females. Males with Dengue had higher occurrence of Bradycardia, CKMB elevations and Echo abnormalities.
- The existence of co infection does not seem to increase the morbidity in this study group.
- The repeat ECG was normal in only 95 patients. Repeat Echo was normal in 99 %.
- Two persons with severe Leptospirosis and one patient with Dengue Shock syndrome died during the study. No deaths occurred in the Malaria group.

CONCLUSION

CONCLUSION

1. This study is first of its kind from Chennai. The observations obtained in this study has been compared with various studies and case reports from other parts of India and rest of the world.
2. Subclinical reversible cardiac involvement is common in Leptospirosis and Dengue while it is less common in Malaria. Cardiac involvement occurs irrespective of the severity of the illness.
3. The mild or nonspecific cardiac symptoms did not correlate with the clinical and laboratory findings, even though giddiness and fatigability were more common in Dengue.
4. Bradycardia and persistant hypotension in Dengue may be clinical markers of underlying myocarditis.
5. Rhythm abnormalities and intra-ventricular conduction disturbances were uncommon in this study population. First Degree Heart blocks are common in Dengue.
6. Reploarisation abnormalities are the commonest ECG finding in three groups. The significance of inferolateral preponderance in leptospirosis and anterolateral preponderance in Dengue has to be evaluated by further studies.
7. CKMB was a relatively sensitive marker for myocarditis in this study. It correlated with the ECG findings and Echo findings in Leptospirosis. Its significance in Malaria is doubtful.
8. Definite echocardiographic evidence of myocarditis was seen in Dengue and Leptospirosis. Reversible valvulitis causing mild or trivial valvular regurgitation was common in Leptospirosis while reversible Global LV hypokinesia was seen in Dengue.

9. Echo evaluation may be recommended for Dengue patiets with bradycardia, CKMB elevations and thrombocytopenia. Tachycardia, ST-T changes and CKMB elevations may warrant Echo evaluation in Leptospirosis.
10. Males are more prone to develop the subclinical cardiac manifestations of Leptospirosis and Dengue whereas females are at risk in Malaria.
11. The cardiovascular manifestations of Leptospirosis, Malaria and Dengue are largely non- fatal, subclinical and reversible in this study conducted on otherwise healthy individuals. Early diagnosis of disease, a well tailored hospital protocol for effective management of disease and presence of less dangerous serovars and serotypes are thought to be the reasons for complete resolution of the changes.
12. This study excluded patients at extermes of age, those with co morbid illnesses, or having other complications of the diseases which may indirectly affect the cardiovascular status. The severity and clinical presentation of cardiac manifestations in the excluded group may vary and has to be evaluated in further studies.

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ANNEXURES

ANNEXURE 1

CASE DEFINITION: MALARIA

Clinical case description:

A case of fever:

May be accompanied with:

- Headache, backache, chills, rigors, sweating, myalgia, nausea and vomiting
- Splenomegaly and anemia
- Generalized convulsions, coma, shock, spontaneous bleeding, pulmonary edema, renal failure and death (untreated falciparum infection)

Laboratory definition of malaria:

Demonstration of malaria parasites in blood films.

Case classification

Suspect case: As per the clinical case definition.

Confirmed case: A suspect case with malaria parasites in blood films.

Confirmed complicated/severe malaria: A confirmed case with symptoms/signs of complicated/severe malaria (prostration, impaired consciousness, respiratory distress (acidotic breathing), multiple convulsions, circulatory collapse, pulmonary oedema (radiological), abnormal bleeding, jaundice, haemoglobinuria, severe anemia, etc).

Confirmed malaria death: Death of a confirmed case.

ANNEXURE 2

CASE DEFINITION: DENGUE FEVER (DF)

Clinical case definition:

An acute febrile illness of 2-7 days duration with 2 or more of the following:

- Headache,
- Retro-orbital pain,
- Myalgia,
- Arthralgia,
- Rash
- Haemorrhagic manifestations
- Leucopoenia

Laboratory criteria for diagnosis:

Any one or more of the following:

• Isolation of the dengue virus from serum, plasma, leukocytes, or autopsy samples

• Demonstration of a fourfold or greater change in reciprocal IgG or IgM antibody titres to one or more dengue virus antigens in paired serum samples (depending on the diagnostic kit used)

• Demonstration of dengue virus antigen in autopsy tissue by immunohistochemistry or immunofluorescence or in serum samples by EIA

• Detection of viral genomic sequences in autopsy tissue, serum or CSF samples by polymerise chain reaction (PCR)

Case classification

Suspected: A case compatible with the clinical description.

Probable: A case compatible with the clinical description with one or more of the following:

• Supportive serology (reciprocal haemagglutination-inhibition antibody titre, comparable IgG EIA titre or positive IgM antibody test in late acute or convalescent-phase serum specimen).

• Epidemiologically linked with a confirmed case of dengue fever (occurrence at same location and time as other confirmed cases of dengue fever).

Confirmed: A case compatible with the clinical description and laboratory confirmed.

Dengue Haemorrhagic Fever (DHF)

A probable or confirmed case of dengue

1. And Hemorrhagic tendencies evidenced by one or more of the following:

- Positive tourniquet test.
- Petechiae, ecchymoses or purpura.
- Bleeding: mucosa, gastrointestinal tract, injection sites or other.
- Haematemesis or melaena.

2. And thrombocytopenia (100,000 platelets or less per mm3).

3. And evidence of plasma leakage due to increased vascular permeability, manifested by one or more of the following:

- >_20% rise in average haematocrit for age and sex.
- >_20% drop in haematocrit following volume replacement treatment compared to baseline.
- Signs of plasma leakage (pleural effusion, ascites, hypoproteinaemia).

Dengue Shock Syndrome (DSS)

All the above criteria, plus evidence of circulatory failure manifested by rapid and weak pulse, and narrow pulse pressure (<_20 mm Hg) or hypotension for age, cold, clammy skin and altered mental status.

ANNEXURE 3

CASE DEFINITION: LEPTOSPIROSIS

Modified Faine's criteria

Clinical features (A)	**Score**	
Fever	**2**	
Headache	**2**	
Temperature > 39 deg.C	**2**	
Myalgia	**4**	
Conjunctival suffusion	**4**	**10**
Meningism	**4**	
Jaundice	**1**	
Albuminuria/ elevated BUN	**2**	
Epidemiological factors (B)		
Rainfall	**5**	
Contaminated environment	**4**	
Animal contact	**1**	
Laboratory criteria (C)		
Culture	**Diagnosis certain**	
ELISA IgM	**15**	
MSAT	**15**	
MAT- single positive high titer	**15**	
MAT- rising titer (paired sera)	**25**	

Each feature is given appropriate scoring.

Presumptive diagnosis of leptospirosis is made of:

- Part A or part A+B with a score of 26 or more
- Part A+B+C = 25 or more and in serological tests, only one test should be scored.

PROFORMA

NAME AGE SEX ADDRESS & LOCALITY

DURATION OF HOSPITAL STAY DURATION OF SYMPTOMS

SYMPTOMS –

Fever	Chills & Rigors	Headache
Nausea & Vomiting	Myalgia	Impaired Consciousness
Conjunctival Suffusion	Jaundice	Oliguria
Shock	Cough	Palpitation
Chest Pain	Bleeding Manifestations	Hemoptysis
Hematuria	Arthralgia	Rash
Retroorbital Pain	Seizures	Calf Tenderness
Facial Flushing	Diarrhea	Abdominal Pain

PHYSICAL EXAMINATION

CONSCIOUSNESS, ORIENTATION, HYDRATION,

PULSE RATE, BLOOD PRESSURE, RESPIRATORY RATE,

TEMPERATURE, O2 SATURATION, PALLOR/ ICTERUS/

CYANOSIS/ CLUBBING/ PEDAL EDEMA/ JVP/ LYMPHADENOPATHY

CVS:

APICAL IMPULSE, HEART SOUNDS, MURMURS,

RS:

RATE, RHYTHM, BREATH SOUNDS, ADDED SOUNDS

ABDOMEN:

TENDERNESS, ORGANOMEGALY ASCITES BOWEL SOUNDS

CNS

FOCAL NEUROLOGIC DEFICITS MENINGEAL SIGNS

INVESTIGATIONS

CBC RUE RFT LFT P.SMEAR MSAT DENGUE IGM QBC CKMB

WIDAL CXR ECG USG ABDOMEN ECHO BT/CT

OTHER TESTS DIAGNOSIS TREATMENT OUTCOME

CONSENT FORM

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு -

மலேரியா, டெங்கு மற்றும் லெப்டோஸ்பைரோசிஸ் ஆகிய நோய்களில் இருதயம் மற்றும் அதன் ரத்த ஓட்டங்களில் ஏற்படும் மாற்றங்களைக் கண்டறியும் ஆய்வு

ஆராய்ச்சி நிலையம் : அரசு ஸ்டான்லி மருத்துவமனை, சென்னை - 600 001.

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் எண் :

பங்கு பெறுவர் இதனை (✓) குறிக்கவும்.

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணிக்கு தெரிவிப்பேன் என உறுதி அளிக்கிறேன். ☐

இந்த ஆய்வில் எனக்கு இரத்தம், சிறுநீர், எக்ஸரே, ஸ்கேன் உட்பட அனைத்து பரிசோதனைகளையும் செய்து கொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன். ☐

பங்கேற்பவரின் கையொப்பம் ..... இடம் ..... தேதி

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

ஆய்வாளரின் பெயர் .....

CERTIFICATE FOR APPROVAL OF ETHICAL COMMITTEE

To

Dr.Prasanth Sankar, PG in MD(GM)

Dear **Dr.Prasanth Sankar, PG in MD(GM)**

The Institutional Ethics Committee reviewed and discussed your application for approval of the project entitled

"Cardiovascular manifestations in patients with leptospirosis, Dengue, Malaria in Chennai "

The following members of the ethics committee were present at the meeting held on 28.01.2010 at the Council Hall, Stanley Medical College, Chennai-1 at 10.00AM

Dr.C.B.Tharani, Director of Pharmacology,
Madras Medical College, Chennai-3 - Chairman of the Ethics Committee
Dr.S. Chitra, Vice-Principal,
Stanley Medical College, Chennai – 1- Member Secretary of the Ethics Committee

MEMBERS

Dr.Jayanthi
Prof.of Medical Gastroenterology
Dr.Madhavan
Prof.of Pharmacology
Dr.E.Dhandapani
Prof.of Medicine
Dr.Sujatha Sridharan
Prof.of Paediatrics
Thiru.Pachaiappan,
Junior Administrative Officer,
Thiru.A. Senthil Manoharan,
Advocate

We approve the project to be conducted in its presented form.

The Institutional Ethics Committee/Independent Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.

Yours sincerely,

Member Secretary,
Ethics Committee

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

ABBREVIATIONS

RWMA	-	Regional Wall Motion Abnormality
MSAT	-	Microscopic Slide Agglutination Test.
MAT	-	Macroscopic Agglutination Test
BUN	-	Blood Urea Nitrogen
AV block	-	Atrioventricular Block
DIC	-	Disseminated Intravascular Coagulation
PCR	-	Polymerase Chain Reaction
ARDS	-	Acute Respiratory Distress Syndrome
AMI	-	Acute Myocardial Infarction
AST	-	Aspartate Transaminase
ALT	-	Alanine Transaminase
CK/CPK	-	Creatine Phosphokinase
CKMB/CPKMB	-	Creatine Phosphokinase MB isoenzyme
EMB	-	Endomyocardial Biopsy
QBC	-	Quantitative Buffy Coat
ESR	-	Erythrocyte Sedimentation Rate

MASTER CHART

NAME	AGE	SEX	HOSPITAL STAY	SYMPTOMS	FEVER	MYALGIA	CALF PAIN	ARTHRALGIA	HEADACHE	CHILLS & RIGORS	COUGH	CHEST PAIN	PALPITATION	GIDDINESS	DYSPNEA	N & V	
AISWARYA	15	F	5	7	1						1						1
AMALA	23	F	3	20	1	1	1			1	1	1	1	1	1	1	1
ANITHA	24	F	5	7	1					1							1
ASHOK	15	M	10	4	1					1	1				1		1
AUGUSTINE	38	M	8	7	1	1	1										1
BABU	49	M	6	12	1	1	1			1	1						1
BASKAR	39	M	5	10	1	1	1			1	1						
BHANUMATHY	15	F	6	6	1										1		1
BOMMI	34	F	6	5	1	1				1		1					1
CHITRA	37	F	5	6	1	1					1						1
DEVA	29	M	7	5	1	1	1			1	1	1					1
DHANABAKIYAM	55	F	6	5	1	1	1										
DHANALAKSHMI	38	F	5	4	1	1					1						1
GANAPATHY	28	M	6	7	1	1				1	1	`					1
GANESHAMURTHY	24	M	6	10	1						1	1					1
GAYATHRI	42	F	5	4	1	1					1						1
GOPAL	52	M	5	10	1	1				1	1						1
GOVIND	17	M	6	7	1	1					1	1				1	1
GOVINDAMMAL	36	F	12	15	1	1	1			1							
HARIPRASAD	17	M	6	3	1						1						1
IBRAHIM	18	M	12	5	1					1							
JABBAR	21	M	15	10	1	1			1	1	1	1			1		1
JANAKIRAMAN	28	M	6	6	1	1					1		1				1
JOHN BASHA	42	M	8	7	1	1	1										1
JOSEPH	15	M	6	8	1					1	1						1
KALAIVANI	13	F	8	6	1	1			1	1	1				1		1
KANNIYAMMAL	32	F	12	15	1	1	1			1							
KARPAGAM	30	F	7	8	1	1			1	1	1	1					1
KARTHIGA	16	F	6	18	1					1	1	1					
KARTHIKA	24	F	7	7	1	1	1			1	1					1	1
KASTHURI	35	F	7	8	1	1			1	1	1	1					1
KISHORE	22	M	10	10	1					1	1						1
KOTHAI	18	F	12	7	1					1							1
KRISHNAVENI	26	F	7	6	1	1							1			1	1
KUPPAN	34	M	6	15	1	1				1	1		1				
LAKSHMI	35	F	10	15	1	1				1	1						1
LALITHA	18	F	3	20	1	1	1			1	1	1	1	1	1	1	1
LEGHA	26	F	7	6	1	1							1			1	1
LOGANATHAN	28	M	9	20	1	1			1	1	1						1
MAHALAKSHMI	13	F	7	7	1												1

RASH	PULSE	SBP	DBP	RR	TEMP	SpO2	PALLOR	JVP	APEX	HS	MURMURS	ADDED SOUNDS	BS	HB1	TC1	DC1	ESR1	PLATELET1	UREA1	CREAT	Na+	K+	BILIRUBIN
	104	100	70	16	100	99	1		N	N			N	10.3	3300	35/61/4	14	81000	38	1	148	4	1
1	140	90	60	28	102	88	1	1	A HYPERI	A LOUD S1 P2	A PSM APEX	1 S3	N	9.5	3800	45/55/0	33	75000	38	0.9	133	3.8	2
	104	120	80	16	99	99			N	N			N	10.8	4000	63/36/1	12	130000	28	0.7	138	3.5	1
	102	90	60	23	100	99			N	N			N	11.6	7000	69/30/1	8	110000	20	0.6	138	3.9	1
	54	110	70	18	98	99			N	N			N	10.8	11200	88/22/0	24	70000	40	1.2	138	3.3	3.8
	88	100	70	22	101	99			N	N			N	11.8	7200	55/45/0	5	100000	18	0.5	137	3.9	3.2
	88	110	80	22	99.8	99			N	N			N	11.8	7200	55/45/0	5	100000	18	0.5	137	3.9	5.6
	100	80	60	20	101	99			N	N			N	11.4	8900	58/43/2	9	130000	25	0.5	141	4	1
1	104	90	70	18	100	99			N	N			N	11	7600	50/46/4	15	150000	24	0.7	144	3.2	0.7
	94	120	80	17	100	98			N	N			N	11.5	7600	80/20/0	8	120000	27	0.9	139	4.1	0.9
	100	130	80	24	101.2	99	1		N	N	PSM APEX		N	10.8	7200	64/35/1	22	120000	22	0.8	141	3.9	0.8
	110	138	80	18	100.1	98	1		N	N			N	8.6	7300	80/20/0	8	130000	32	1	135	4.1	0.9
	98	110	80	16	99.8	99			N	N			N	10.8	9200	70/30/0	12	160000	15	0.6	140	4	0.8
	112	120	80	18	99.5	99			N	N			N	10.6	7100	30/70/0	15	180000	24	0.8	144	3.9	1
	75	110	70	18	98.4	99			N	N			N	12.8	6900	68/32/0	10	100000	29	0.8	140	3.9	1
	94	110	80	16	99.8	99			N	N			N	10.8	9200	70/30/0	12	160000	15	0.6	140	4	0.8
	76	110	70	18	99.6	99			N	N			N	10.4	5200	60/37/3	12	120000	30	0.7	139	4.2	0.8
	110	90	60	26	100	99			N	N			N	11.8	6700	67/33/0	7	100000	29	0.6	140	3.8	0.8
	84	120	80	18	100	99			N	N			N	10.5	9400	55/43/2	20	180000	26	0.6	136	4.1	0.9
	112	100	60	22	100.6	99			N	N			N	12	7400	85/15/0	7	130000	30	0.9	137	3.7	0.8
	54	100	70	18	100.6	99	1		N	N		1 S3	N	10.3	2700	54/40/6	25	43000	29	0.9	146	4.5	0.9
	48	90	70	20	100	98		1	N	A MUFFLED		1 S3	N	13.7	3500	45/52/3	7	70,000	27	0.8	126	3.6	0.9
	102	130	90	16	101.2	99			N	N			N	11.5	6300	43/55/2	7	160000	43	1.2	138	3.4	0.8
	56	110	70	18	98	99			N	N			N	10.8	11200	88/22/0	24	70000	40	1.2	138	3.3	3.8
	110	90	60	22	101	98	1		N	N			N	10.2	4700	61/35/2	10	110000	34	0.8	138	3.6	0.9
	106	80	60	20	101	98	1		N	N			N	10.2	6900	68/32/0	15	120000	24	0.7	139	3.5	0.9
	80	120	80	18	100	99			N	N			N	10.5	9400	55/43/2	20	180000	26	0.6	136	4.1	0.9
	104	120	80	24	104	98			N	N			N	10.1	6000	62/36/2	20	132000	22	0.8	139	3.2	0.9
	120	110	60	20	101	99	1		N	N		1 S3	N	8.9	8000	40/60/0	17	250000	23	0.7	141	4	0.9
	114	110	70	26	102	99			N	N			N	12	10000	80/18/2	23	180000	29	0.9	139	4.4	0.9
	102	120	80	24	104	98			N	N			N	10.1	6000	62/36/2	20	132000	22	0.8	139	3.2	0.9
	80	100	70	18	100	99			N	N			D LUNG BASES	14.8	3200	61/36/3	5	52000	24	0.9	139	3.6	0.7
	76	110	70	16	100.2	99	1		N	N			N	10.5	3100	55/45/0	23	37000	28	0.9	139	3.8	1
	90	100	70	20	100	99	1		N	N			N	10	4000	35/65/0	26	49000	39	1.3	130	3.3	1
	75	90	60	16	99.8	99			N	N			N	12.2	7600	40/56/4	12	140000	17	0.8	133	3.4	0.8
	112	100	60	18	101.4	99			N	N			N	11.8	9800	55/44/1	18	120000	28	1	146	4.2	0.8
1	134	90	60	28	102	88	1	1	A HYPERI	A LOUD S1 P2	A PSM APEX	1 S3	N	9.5	3800	45/55/0	33	75000	38	0.9	133	3.8	2
	96	100	70	20	100	99	1		N	N			N	10	4400	30/67/3	25	46000	40	1.2	135	3.6	0.9
	76	130	80	18	98.6	99			N	N			N	11.2	6200	65/30/5	15	150000	25	0.8	136	3.6	0.8
	104	110	70	24	101	99			N	N			N	11.2	6000	80/20/0	12	150000	22	0.7	126	3.3	0.8

P.SMEAR/QB	MSAT	DENGUE IGM	CK-T	CKMB	INDEX	CXR	ECG-1 RATE	RHYTHM	P	PR	QRS	ST	T	ECG-2RATE	RHYTHM	P	PR	ORS	ST
1F			88	12	13.63	N	112	N	N	N	N	N	N	75	N	N	N	N	N
N	1		1126	324	28.77	A - PUL EDEMA	140	N	N	D	N	D 1,2,3,L,F,V1-V6	I 1,2,3,L,F,V1-V6	120	N	N	D	N	D 1,2,3,L,F,V
N	1		165	30	18.8	N	100	N	N	N	N	N	N	68	N	N	N	N	N
N		1	320	26	8.12	N	68	I ATRIAL ECTOPIC	A 2,3,F P PU	N	N	N	N	80	N	N	N	N	N
N	1		300	98	32.57	N	55	N	N	N	A LOW VOLTA	N	N	80	N	N	N	N	N
N	1		150	66	44	N	88	N	N	N	N	N	N	74	N	N	N	N	N
N	1		48	37		N	88	N	N	N	N	N	N	74	N	N	N	N	N
1F		1	80	31		N	104	N	N	N	N	D 2,3,F	1 V1-V3	75	N	N	N	N	N
N		1	1400	120	8.56	N	125	N	N	N	N	D V1-V6	I V1-V5	90	N	N	N	N	N
1F			89	20	22.47	N	75	N	N	N	N	N	I V1-V6	70	N	N	N	N	N
N	1	1	357	100	28.01	N	100	N	N	N	N	N	N	72	N	N	N	N	N
1V			98	66	67.34	N	115	N	N	N	N	D 1.Vl,V5,V6	I 1,Vl,V5,V6	88	N	N	N	N	N
V			120	13	10.83	N	115	N	N	N	N	N	N	72	N	N	N	N	N
N		1	266	19	7.14	N	115	N	N	N	N	N	N	72	N	N	N	N	N
N		1	97	10	10.3	N	80	N	N	N	N	N	N	70	N	N	N	N	N
V			120	13	10.83	N	115	N	N	N	N	N	N	72	N	N	N	N	N
N	1		196	44	22.44	N	80	N	N	N	N	ERS V3-V6	N	90	N	N	N	N	N
1V			357	55	15.4	N	114	N	N	N	N	N	N	78	N	N	N	N	N
N	1		117	18	15.38	N	80	N	N	N	N	N	N	80	N	N	N	N	N
1V			97	10	10.3	N	115	N	N	N	N	N	N	75	N	N	N	N	N
N		1	253	76	30.03	N	46	N	N	D	N	N	I V3-V6	96	N	N	D	N	N
N		1	788	156	19.44	N	46	N	N	I	N	ERS V3-V6	N	60	N	N	N	N	ERS V3-V6
1V			52	26		N	130	N	N	N	N	N	N	80	N	N	N	N	N
N	1		300	98	32.57	N	55	N	N	N	A LOW VOLTA	N	N	80	N	N	N	N	N
N	1		120	55	45.83	N	115	N	N	N	N	N	I 1,L,V3-V6	75	N	N	N	N	N
1F			2205	94	4.26	N	110	N	N	N	N	N	N	80	N	N	N	N	N
N	1		117	18	15.38	N	80	N	N	N	N	N	N	80	N	N	N	N	N
1V			69	40		N	114	N	N	N	N	N	I 2,3,F,V1-V6	96	N	N	N	N	N
1F			198	27	13.63	N	140	N	N	N	N	N	N	78	N	N	N	N	N
N	1		1322	74	5.55	N	116	N	N	N	N	N	N	70	N	N	N	N	N
1V			71	38		N	114	N	N	N	N	N	I 2,3,F,V1-V6	96	N	N	N	N	N
N		1	54	29		A BL PE	75	N	N	N	N	N	N	80	N	N	N	N	N
N		1	256	87	33.9	A L PE MIN	75	N	N	N	N	N	N	80	N	N	N	N	N
1V		1	135	22	16.29	N	96	N	N	N	N	N	N	90	N	N	N	N	N
N		1	87	15	17.24	N	75	N	N	N	N	ERS V2-V6,1,L	N	80	N	N	N	N	N
1V			104	35	33.6	N	115	N	A LAA	N	N	N	I V4-V6	80	N	LAA	N	N	N
N	1		1078	376	34.87	A - PUL EDEMA	140	N	N	D	N	D 1,2,3,L,F,V1-V6	I 1,2,3,L,F,V1-V6	120	N	N	D	N	D 1,2,3,L,F,V
N		1	138	24	16.33	N	96	N	N	N	N	N	N	90	N	N	N	N	N
1V			116	28	24.13	N	75	N	N	N	N	N	N	70	N	N	N	N	N
N	1		129	35	27.1	N	100	N	N	N	N	N	N	80	N	N	N	N	N

T	ECHO1	ECHO2	LEPTO	MALARIA	DENGUE	OUTCOME
N	N	NA		1		DISCHARGE
I 1,2,3,L,F,V1-\	A EF 38 MILD MR	NA	1			DEATH
N	N	NA	1			DISCHARGE
N	N	NA			1	DISCHARGE
N	A EF 40	N	1			DISCHARGE
I V1	N	NA	1			DISCHARGE
I V1	N	NA	1			DISCHARGE
N	N	NA		1	1	DISCHARGE
N	N	NA			1	DISCHARGE
I V1-V3	N	NA		1		DISCHARGE
N	A MILD MR	N	1		1	DISCHARGE
N	N	NA		1		DISCHARGE
N	N	NA		1		DISCHARGE
N	N	NA			1	DISCHARGE
N	N	NA			1	DISCHARGE
N	N	NA		1		DISCHARGE
N	A TRIVIAL TR	N	1			DISCHARGE
N	N	NA		1		DISCHARGE
N	N	NA	1			DISCHARGE
N	N	NA		1		DISCHARGE
N	A EF 46	N			1	DISCHARGE
N	A EF 40	N			1	DISCHARGE
N	N	NA		1		DISCHARGE
N	A EF 40	N	1			DISCHARGE
N	N	NA	1			DISCHARGE
N	N	NA		1		DISCHARGE
N	N	NA	1			DISCHARGE
I 2,3,F,V1-V6	A TRIVIAL MR	N		1		DISCHARGE
N	N	NA		1		DISCHARGE
N	N	NA	1			DISCHARGE
I 2,3,F,V1-V6	A TRIVIAL MR	N		1		DISCHARGE
N	N	NA			1	DISCHARGE
N	A MINIMAL PE	N			1	DISCHARGE
N	N	NA		1	1	DISCHARGE
N	N	NA			1	DISCHARGE
N	N	NA		1		DISCHARGE
I 1,2,3,L,F,V1-\	A EF 38 MILD MR	NA	1			DEATH
N	N	NA			1	DISCHARGE
N	N	NA		1		DISCHARGE
I V1-V3	N	NA	1			DISCHARGE

MAHESH	45	M	9	6	1	1			1			1			1			1	1	1
MARIYAMMAL	15	F	9	20	1					1		1	1					1		1
MATHI	27	F	20	14	1	1							1							1
MEENAKSHI	22	F	12	7	1	1				1										1
MIROSH	18	M	14	9	1	1			1	1		1	1					1		1
MOORTHY	27	M	6	6	1	1		1				1	1							1
MUNIYAMMAL	41	F	5	3	1				1			1								
MURUGAMMAL	35	F	8	6	1	1			1	1		1						1		1
MURUGAN	16	M	5	7	1	1				1		1							1	
MUTHU	13	M	10	4	1					1		1								1
OTHAM	21	M	15	5	1					1										
PENICILLAMMAL	38	F	5	3	1				1			1								
PERIYASAMI	44	M	5	6	1	1				1		1								1
PIYUS	24	M	15	5	1					1										
PRABESH	24	M	10	10	1	1			1	1										
PRABHU	23	M	9	5	1	1						1	1		1				1	1
PRADIP	29	M	10	10	1	1			1	1										
PRASAD	35	M	14	6	1					1									1	1
PREMA	13	F	9	7	1	1				1			1							1
RAFIQ	50	M	10	14	1	1			1	1		1								1
RAJA	32	M	7	4	1	1		1		1		1			1					1
RAJALAKSHMI	28	F	10	12	1	1		1		1		1								
RAJATJHI	20	F	6	5	1	1				1			1							1
RAJESWARI	21	F	6	3	1					1		1								1
RAJESWARI	28	F	10	12	1	1		1		1		1								
RAMAKRISHNAN	17	M	6	7	1	1						1	1						1	1
RAVINDAR	23	m	10	15	1								1							1
ROBERT	49	M	6	7	1	1				1										1
SAMPATH	31	M	10	5	1	1				1		1								1
SAMPATH KUMAR	43	M	10	3	1	1		1		1		1					1		1	
SATHYA	13	F	5	8	1				1			1						1		1
SEKAR	28	M	6	15	1	1				1		1			1					
SENGAMMAL	55	F	7	10	1							1					1			
SHAFIULLA	46	M	6	15	1	1			1	1		1								1
SHANTHAKUMARI	14	F	9	20	1					1		1	1							1
SHOBHANA	18	F	6	6	1													1		1
SIMON	45	M	6	7	1	1				1										1
SRINIVAS	24	M	7	7	1	1		1		1		1							1	1
SRINIVASAN	25	m	10	15	1								1							1
SULTHAN	48	M	10	14	1	1			1	1		1								1
SURESH	24	m	10	5	1	1				1		1								1

1	50	80	60	26	101	95	1	N	N		1 S3	D LUNG BASES	9.8	3600	40/58/2	15	39000	40	1.1	136	3	1.2
	112	90	70	20	101	98	1	N	N			N	10.2	4200	80/18/2	16	150000	30	0.6	134	3.4	1
1	96	110	80	18	100	99	1	N	N			N	9.8	13,700	85/14/1	10	190000	30	0.6	139	3.9	1
1	108	100	70	20	99	98		N	N			N	11	3800	55/43/2	25	33000	45	1.4	135	3.5	1
	46	90	60	18	99	99		1 N	A MUFFLED		1 S3	N	13.7	3500	45/52/3	7	70,000	27	0.8	126	3.6	0.9
	58	110	80	16	99	99		N	N			N	14.2	4800	58/40/2	13	120000	24	0.7	136	2.8	0.9
	84	110	80	18	99.8	99		N	N			N	10.8	10500	62/35/3	15	140000	20	0.7	141	4	0.8
	110	80	60	20	101	98	1	N	N			N	10.2	6900	68/32/0	15	120000	24	0.7	139	3.5	0.9
	102	120	80	24	101.3	99		N	N			N	13	6100	86/14/0	5	60,000	28	0.7	140	3.3	0.8
	104	90	60	23	100	99		N	N			N	11.6	7000	69/30/1	8	110000	20	0.6	138	3.9	1
	102	120	80	24	101	96		N	N			N	11	7000	60/40/0	12	100000	24	0.6	132	3.1	1
	88	110	80	18	99.8	99		N	N			N	10.8	10500	62/35/3	15	140000	20	0.7	141	4	0.8
	110	100	70	23	100.5	98		N	N		1 S3	N	11.2	4000	70/28/2	25	70000	20	0.7	138	3.8	1
	100	120	80	24	101	96		N	N			N	11	7000	60/40/0	18	110000	33	0.6	132	3.1	1
1	50	100	60	26	102	99	1	N	N			N	13	4700	61/35/4	10	50000	30	0.8	140	3.8	1
	124	120	80	26	102	99		N	N	PSM APEX	1 S3	N	11	5600	48/50/2	7	100000	21	1.3	131	3.9	0.9
1	80	120	80	26	102	99	1	N	N			N	13	4700	61/35/4	10	50000	30	0.8	140	3.8	1
1	52	100	60	16	100	99		N	A MUFFLED			D LUNG BASES	14.3	4200	61/34/5	45	19000	29	0.8	144	4	1.5
	130	100	70	26	100.8	97	1	N	N			N	10.8	6000	60/40/0	12	130000	28	0.9	138	4.1	0.8
	122	110	70	24	101	99		N	N			N	12	6800	66/34/0	12	100000	34	1.1	135	3.6	2
	110	100	70	22	100.5	98		N	N			N	14.8	5900	55/45/0	7	100000	24	0.8	140	4.3	1
	108	100	60	22	101	97	1	N	N			N	10	6200	62/38/0	7	100000	30	0.7	135	3.4	0.8
1	102	90	70	18	100	99		N	N			N	11	7600	50/46/4	15	150000	24	0.7	144	3.2	0.7
	98	120	80	18	99.4	99		N	N			N	10.2	4000	50/46/4	35	45000	26	0.7	138	4	1
	110	100	60	22	101	97	1	N	N			N	10.2	7800	62/35/3	12	100000	30	0.8	139	3.4	0.8
	114	90	60	26	100	99		N	N			N	11.8	6700	67/33/0	7	100000	29	0.6	140	3.8	0.8
	88	110	70	18	99	99		N	N			N	12.6	11200	60/40/0	12	100000	24	0.9	140	4	1
	104	100	70	16	99	98		N	N			N	13	4500	68/30/2	11	150000	15	0.8	145	2.5	1
	108	120	70	18	100	99	1	N	N			N	10.5	7000	80/20/0	15	120000	63	1.3	150	3.4	1.1
	110	100	60	24	101	98		N	N		1 S3	N	12.4	10,300	50/46/4	8	240000	33	1.1	138	3.5	0.9
	96	80	50	22	98	99		N	N			N	12.6	5000	65/35/0	12	120000	22	0.6	140	4	0.9
	78	90	60	16	99.8	99		N	N			N	12.2	7600	40/56/4	12	140000	17	0.8	133	3.4	0.8
	98	120	70	18	100	99	1	N	A IRR IRR			N	10	10800	80/18/2	19	230000	34	0.9	143	4	0.7
	86	110	60	18	101	99		N	N			N	11.2	6600	70/29/1	5	100000	26	0.9	141	3.7	1.3
	116	90	70	20	101	98	1	N	N			N	10.2	4000	80/20/0	14	140000	28	0.6	133	3.4	1
	104	80	60	20	101	99		N	N			N	11.4	8900	58/43/2	9	130000	25	0.5	141	4	1
	108	100	70	16	99	98		N	N			N	13	4500	68/30/2	11	150000	15	0.8	145	2.5	1
	112	110	70	26	102	99		N	N			N	12.6	11500	88/12/0	15	150000	28	1	144	4.2	0.9
	86	110	70	18	99	99		N	N			N	12.6	11200	60/40/0	12	100000	24	0.9	140	4	1
	120	110	70	24	101	99		N	N			N	12	6800	66/34/0	12	100000	34	1.1	135	3.6	2
	100	120	70	18	100	99	1	N	N			N	10.5	7000	80/20/0	15	120000	63	1.3	150	3.4	1.1

N		1	285	81	28.42	A - B/L PL. EFFU	50	N	N	I 1HB	N	D V4-V6	I V4-V6	54	N	N	N	D 1,2,3,L,F,V
N	1	1	92	15	11.32	N	120	N	N	N	N	D V1-V6	I V1-V5	75	N	N	N	N
N		1	65	43		N	84	N	N	N	N	D 2,3,F,V1-V5	I 2,3,F,V1-V4	78	N	N	N	N
N		1	255	45	17.64	N	80	N	N	N	N	N	N	84	N	N	N	N
N		1	782	152	19.43	N	46	N	N	I	N	ERS V3-V6	N	60	N	N	N	ERS V3-V6
N	1		67	13		N	57	N	N	N	N	N	I 1,L,V3-V6	60	N	N	N	N
1V			88	57	64.77	N	88	N	N	N	N	N	I V1-V4	80	N	N	N	N
1F			2264	99	4.26	N	110	N	N	N	N	N	N	80	N	N	N	N
1V			386	91	23.57	N	125	N	N	I	N	N	N	80	N	N	N	N
N		1	320	26	8.12	N	68	I ATRIAL ECTOPIC	A 2,3,F P PU	N	N	N	N	80	N	N	N	N
N		1	235	98	41.7	N	114	N	N	N	IRBBB	N	N	84	N	N	N	N
1V			88	57	64.77	N	88	N	N	N	N	N	I V1-V4	80	N	N	N	N
1F			327	17	5.19	N	114	N	N	N	N	D 2,3,F,V1-V6	I 2,3,F,V1-V6	77	I -SA NODAL DIS	N	N	N
N		1	235	98	41.7	N	114	N	N	N	IRBBB	N	N	84	N	N	N	N
N	1	1	289	89	30.79	N	54	N	N	N	N	N	I V1-V4	60	N	N	N	N
N	1		278	91	32.85	N	150	N	N	N	N	D 2,3,F,V3-V6	I 2,3,F,V2-V6	75	N	N	N	N
N	1	1	103	30	29.12	N	83	N	N	N	N	N	I V1-V6	75	N	N	N	N
N		1	378	100	26.45	A BL PE	38	N	N	N	A LOW VOLTA	N	N	60	N	N	N	N
1V			129	25	19.37	N	150	N	N	N	N	D 2,3,F,V2-V6	I 2,3,F,V2-V6	72	N	N	N	N
N		1	100	24	24	N	120	N	N	N	N	N	N	75	N	N	N	N
N	1		712	73	10.25	N	115	N	A 2,3,F P PU	N	A LAFB	D V3-V6	I V3-V6	68	N	N	N	N
1V	1		301	98	32.55	N	108	N	N	N	N	N	I 2,3,F,V4-V6	75	N	N	N	N
N		1	1401	115	8.21	N	125	N	N	N	N	D V1-V6	I V1-V5	90	N	N	N	N
N	1		658	59	8.96	N	108	N	N	N	N	D V4-V6	I V4-V6	80	N	N	N	N
1V	1		298	94	32.11	N	104	N	N	N	N	N	I 2,3,F,V4-V6	80	N	N	N	N
1V			357	55	15.4	N	114	N	N	N	N	N	N	78	N	N	N	N
N	1		765	112	14.64	N	100	N	N	D	IRBBB	N	N	70	N	N	D	N	N
1V			77	10		N	75	N	N	N	N	N	N	80	N	N	N	N	N
1V			78	20		N	100	N	N	N	N	N	N	75	N	N	N	N	N
N	1		288	78	27.08	N	150	N	N	N	N	D 2,3,F,V3-V6	I 2,3,F,V3-V6	69	N	N	N	N	N
N		1	85	10	11.76	N	80	N	N	N	N	D 2,3,F,V1-V4	I 2,3,F,V1-V4	80	N	N	N	N	N
N		1	87	15	17.24	N	75	N	N	N	N	ERS V2-V6,1,L	N	80	N	N	N	N	N
N	1		800	88	11	N	100	I VENT ECTOPIC	N	N	N	D 2,3,F	I 2,3,F	75	N	N	N	N	N
N		1	88	57	64.77	N	78	N	N	N	N	N	N	88	N	N	N	N	N
N	1	1	90	10	11.11	N	125	N	N	N	N	D V1-V6	I V1-V5	80	N	N	N	N	N
N		1	79	31		N	104	N	N	N	N	D 2,3,F	1 V1-V3	75	N	N	N	N	N
1V			77	10		N	75	N	N	N	N	N	N	80	N	N	N	N	N
N	1		1224	70	5.7	N	112	N	N	N	N	N	N	75	N	N	N	N	N
N	1		765	112	14.64	N	100	N	N	D	IRBBB	N	N	70	N	N	D	N	N
N		1	100	24	24	N	120	N	N	N	N	N	N	75	N	N	N	N	N
1V			78	20		N	100	N	N	N	N	N	N	75	N	N	N	N	N

I 1,2,3,L,F,V1-\	A EF 50 MILD PE	EF 46 MILD PE				1	DEATH
N	A TRIVIAL MR	N	1			1	DISCHARGE
N	A TRIVIAL TR	N				1	DISCHARGE
N	N	NA				1	DISCHARGE
N	A EF 40	N				1	DISCHARGE
N	N	NA	1				DISCHARGE
N	N	NA		1			DISCHARGE
N	N	NA		1			DISCHARGE
N	N	NA		1			DISCHARGE
N	N	NA				1	DISCHARGE
N	A EF 40	N				1	DISCHARGE
N	N	NA		1			DISCHARGE
N	A MILD TR	N		1			DISCHARGE
N	A EF 40	N				1	DISCHARGE
N	A TRIVIAL MR	N	1			1	DISCHARGE
N	A MILD MR	N	1				DISCHARGE
N	N	NA	1			1	DISCHARGE
N	A EF 40 MILD PE	N				1	DISCHARGE
N	N	NA		1			DISCHARGE
N	N	NA				1	DISCHARGE
N	A MR TRIVIAL	N	1				DISCHARGE
N	N	NA	1	1			DISCHARGE
N	N	NA				1	DISCHARGE
N	N	NA	1				DISCHARGE
N	N	NA	1	1			DISCHARGE
N	N	NA		1			DISCHARGE
N	N	NA	1				DISCHARGE
N	N	NA		1			DISCHARGE
N	N	NA		1			DISCHARGE
N	N	NA	1				DISCHARGE
N	N	NA				1	DISCHARGE
N	N	NA				1	DISCHARGE
N	N	NA	1				DISCHARGE
N	A TRIVIAL MR	N				1	DISCHARGE
N	A TRIVIAL MR	N	1			1	DISCHARGE
N	N	NA				1	DISCHARGE
N	N	NA		1			DISCHARGE
N	N	NA	1				DISCHARGE
N	N	NA	1				DISCHARGE
N	N	NA				1	DISCHARGE
N	N	NA		1			DISCHARGE

TAUFIQ	45	M	6	15	1	1			1	1	1							1
UMAPATHY	27	M	9	20	1	1			1	1	1							1
VALLI	20	F	12	7	1	1				1								1
VASUGI	17	F	12	7	1					1								1
VEDHA	31	F	10	15	1	1				1	1							1
VENKATESAN	33	M	8	7	1				1		1				1			1
VENUGOPAL	52	M	5	10	1	1				1	1							1
vigraman	15	M	5	7	1	1				1	1						1	
VIJAY	19	M	10	6	1	1				1	1	1						1
VIJAYALAKSHMI	17	F	9	20	1					1	1	1						1
VIKRAMAN	35	M	14	6	1					1							1	1
VIMAL	32	M	7	5	1	1	1			1	1	1						1
VINAYAGAM	29	M	6	7	1	1				1	1	`						1
VINITHA	30	F	5	7	1					1								1
VINOTH	40	M	6	12	1	1	1			1	1							1
VISHNU	24	M	7	7	1	1	1			1	1						1	1
VISWANATHAN	14	M	5	6	1	1				1	1							1
VIVETHA	15	F	5	7	1						1							1
WILSON	52	M	6	7	1	1				1								1

	90	110	60	18	101	99		N	N			N	11.2	6600	70/29/1	5	100000	26	0.9	141	3.7	1.3
	80	130	80	18	98.6	99		N	N			N	11.2	6200	65/30/5	15	150000	25	0.8	136	3.6	0.8
1	100	100	70	20	99	98		N	N			N	11	3900	55/43/2	25	30000	45	1.4	135	3.5	1
	72	110	70	16	100.2	99	1	N	N			N	10.5	3100	55/45/0	23	37000	28	0.9	139	3.8	1
	110	100	60	18	101.4	99		N	N			N	11.8	9800	55/44/1	18	120000	28	1	146	4.2	0.8
1	52	90	60	26	102	97	1	N	N		1 S3	D LUNG BASES	10.9	4100	40/58/2	15	35000	40	1.1	136	3	1.3
	82	110	70	18	99.6	99		N	N			N	10.4	6000	64/33/3	16	130000	25	0.6	141	4	0.8
	104	120	80	24	101.3	99		N	N			N	13	6100	86/14/0	5	60,000	28	0.7	140	3.3	0.8
	80	96	70	18	100	99		N	N			N	10.8	4000	57/40/3	25	100000	26	0.9	140	3.9	1
	110	90	70	20	101	98	1	N	N			N	10.2	4000	80/20/0	14	140000	28	0.6	133	3.4	1
1	50	100	60	16	100	99		N	A MUFFLED			D LUNG BASES	14.3	4200	61/34/5	45	19000	29	0.8	144	4	1.5
	100	90	70	20	102	98		N	N			N	10	7900	59/38/1	40	63,000	25	0.8	138	4	1
	110	120	80	18	99.5	99		N	N			N	10.6	7100	30/70/0	15	180000	24	0.8	144	3.9	1
	100	120	80	16	99	99		N	N			N	10.8	4000	63/36/1	12	130000	28	0.7	138	3.5	1
	90	100	70	22	101	99		N	N			N	11.8	7200	55/45/0	5	100000	18	0.5	137	3.9	3.2
	114	110	70	26	102	99		N	N			N	13	12000	82/16/2	15	150000	28	0.7	140	4.2	0.9
	114	100	70	23	100.5	98		N	N		1 S3	N	11.2	4000	70/28/2	25	70000	20	0.7	138	3.8	1
	106	100	70	16	100	99	1	N	N			N	10	3000	38/60/2	22	75000	32	0.9	140	4.2	0.7
	100	100	70	16	99	98		N	N			N	13	4500	68/30/2	11	150000	15	0.8	145	2.5	1

N		1	88	57	64.77	N	78	N	N	N	N	N	N	88	N	N	N	N	N
1V			116	28	24.13	N	75	N	N	N	N	N	N	70	N	N	N	N	N
N		1	255	45	17.64	N	80	N	N	N	N	N	N	84	N	N	N	N	N
N		1	256	87	33.9	A L PE MIN	75	N	N	N	N	N	N	80	N	N	N	N	N
1V			104	35	33.6	N	115	N	A LAA	N	N	N	I V4-V6	80	N	LAA	N	N	N
N		1	180	78	43.33	A - B/L PL. EFFU	52	N	N	I 1HB	N	N	N	60	N	N	N	N	N
N	1		200	46	23.1	N	70	N	N	N	N	ERS V3-V6	N	85	N	N	N	N	N
1V			386	91	23.57	N	125	N	N	I	N	N	N	80	N	N	N	N	N
1F			1049	51	4.99	N	80	N	N	N	N	N	N	80	N	N	N	N	N
N	1	1	90	10	11.11	N	125	N	N	N	N	D V1-V6	I V1-V5	80	N	N	N	N	N
N		1	378	100	26.45	A BL PE	38	N	N	N	A LOW VOLTA	N	N	60	N	N	N	N	N
N	1		120	58	48.33	N	107	N	N	N	N	D 2,3,F	N	90	N	N	N	N	N
N		1	266	19	7.14	N	115	N	N	N	N	N	N	72	N	N	N	N	N
N	1		165	30	18.8	N	100	N	N	N	N	N	N	68	N	N	N	N	N
N	1		150	66	44	N	88	N	N	N	N	N	N	74	N	N	N	N	N
N	1		1024	60	5.85	N	108	N	N	N	N	N	N	80	N	N	N	N	N
1F			327	17	5.19	N	114	N	N	N	N	D 2,3,F,V1-V6	I 2,3,F,V1-V6	77	I -SA NODAL DIS	N	N	N	N
1F			92	14	13.93	N	116	N	N	N	N	N	N	70	N	N	N	N	N
1V			77	10		N	75	N	N	N	N	N	N	80	N	N	N	N	N

N	A TRIVIAL MR	N			1	DISCHARGE
N	N	NA		1		DISCHARGE
N	N	NA			1	DISCHARGE
N	A MINIMAL PE	N			1	DISCHARGE
N	N	NA		1		DISCHARGE
N	A EF 48	N			1	DISCHARGE
N	A TRIVIAL TR	N	1			DISCHARGE
N	N	NA		1		DISCHARGE
N	A EF 50 TRIVIAL TR	N		1		DISCHARGE
N	A TRIVIAL MR	N	1		1	DISCHARGE
N	A EF 40 MILD PE	N			1	DISCHARGE
N	N	NA	1			DISCHARGE
N	N	NA			1	DISCHARGE
N	N	NA	1			DISCHARGE
I V1	N	NA	1			DISCHARGE
N	N	NA	1			DISCHARGE
N	A MILD TR	N		1		DISCHARGE
N	N	NA		1		DISCHARGE
N	N	NA		1		DISCHARGE